BERIBERI IN FIJI, 1894-5

1896.

Legislative Council.

FIJI.

COUNCIL PAPER, No. 1.

EPIDEMIC OF BERIBERI.

(OCCURRING AMONG JAPANESE IMMIGRANTS IN 1894-5.—REPORT ON.)

Laid on the Table, 7 *February*, 1896.

SCHEDULE.

No. 1.

Minute

By The Honourable the Chief Medical Officer to The Honourable the Acting Colonial Secretary.

The nature of the epidemic described in the two Reports which I have the honour to transmit herewith for the information of the Governor—and the fact that this was the first known occurrence of Beriberi in this Colony in its epidemic form—have seemed to me to call for some general account of the disease as it has been met with in other countries, for His Excellency's further satisfaction.

2. The accompanying Reports, by the District Medical Officers at Rewa and Labasa respectively, not merely present in an exceptionally clear and succinct form a history of the two subdivisions of the epidemic as it occurred under their observation, but they may be fairly said to pourtray the sum of our modern clinical knowledge of this exotic disease. And while they afford useful corroborative evidence

evidence on most of the views recorded by other observers during the last few years, they help generally to elucidate the essential points concerning Beriberi, which until quite lately was regarded as a very obscure disease; and about which but few medical men, excepting those who have come into contact with it abroad, even now know anything at all.

3. The accounts published concerning Beriberi in so far as they depend upon original research are for the most part fragmentary, and far between. The most pointed and complete description of it is the work of Drs. Pekelharing and Winkler, of the University of Utrecht, who were directed by the Netherlands Government to carry out its investigation in Sumatra, in consequence of the great extent to which the Dutch troops were infected by it during the Atjeh war. The results of their labours were published in Dutch,* and have been translated into English, French, and German—if not also Portuguese—and form, now, the standard reference on the subject.

An excellent monograph on Beriberi is also to be found in Davidson's "Hygiene and Diseases of Warm Climates,"† written by Dr. Patrick Manson, which affords a summary of modern knowledge of the disease; and this will probably be the text-book for English students of tropical medicine for some time to come.

4. Beriberi may be described as a specific breakdown of the motor and sensory nerves (principally), usually manifesting its effects first about their peripheral portions but extending later to the more central tracts, and involving muscles and organs of vital importance which in health are regulated by the sympathetic system. In the latter way it tends to cause death, by paralysing the heart and the muscles of breathing, or by precluding the continuance of the digestive and assimilative functions; or by choking, so to call it, the lungs and other viscera by means of the œdema to which, in one class of cases, it gives rise.

5. This type is, for convenience of description, called 'Wet Beriberi,' and is characterised by dropsical distension (œdema): the other, the atrophic or 'Dry' form, leads to rapid and extreme wasting of the body. In both the patient becomes paralysed and helpless; and there is a tendency to terminate suddenly in death in the manner described. The photographs taken by Dr. Joynt and submitted with these Reports, show very strikingly the appearances of persons (Japanese) suffering from the 'Wet,' and the 'Dry,' form of Beriberi respectively.‡

6. In some parts of the world Beriberi is an endemic disease; but it also frequently assumes the epidemic form. Japan, where it is known as 'Kak'kè' (a term now often used also by European physicians), is one of those countries.

It is stated on excellent authority§ that "there is evidence in the writings of the physicians of China and Japan that Beriberi has existed in these countries from remotest antiquity." I have found it mentioned in the narrative‖ given by Dr. G. F. Gemelli Careri, an Italian traveller, who made the voyage from Manila to Acapulco

* Onderzoek naar den aard en de oorzaak der Beriberi, en de Middelen om de Ziekte to Bestrijden. (An investigation into the nature and origin of Beriberi, and the means for subduing this disease).—Utrecht, 1888.

† London and Edinburgh, 1893.

‡ (Photographs not reproduced.)

§ Patrick Manson in "Hygiene and Diseases of Warm Climates," edited by Andrew Davidson, M.D.—1893.

‖ Giro del mondo,—G. F. Gemelli Careri.—Napoli, 1699.

Acapulco in a Spanish galleon, in the latter half of the seventeenth century; and who draws distinctions between it and scurvy and even diagnosed the presence of both these diseases together in the person of the chief pilot, who was a Portuguese born in Madeira. I also find Beriberi alluded to by Ribeyro, the historian of the colonisation of Ceylon, writing in 1685, in these terms (French translation from the Portuguese) :—" Il y a une autre maladie que ceux du pays appellent *Beré bere*, " et à laquelle les Portugais sont fort sujets; c'est une espece de crampe, mais si " violente, que ceux qui en sont attaquez tombent par terre, et on couperoit par " morceaux la partie malade, qu'ils ne le sentiroient pas. Le meilleur remede est de " manger de la chair de porc et du biscuit, de boire du vin de palmier, et de fumer; " on n'a pas vêcu ainsi trois ou quatre mois, qu'on est entierement gueri."*—(*Sic.*)

From this we not only note the prevalence of Beriberi in Ceylon in the seventeenth century, under the same name as to-day, but we see also that the necessity for a duly nitrogenous diet, an antiscorbutic drink, and microbicide fumigation, which together constitute the modern prophylactic measures advised, were already known and popular.

As regards India it does not appear that any references to Beriberi in its ancient literature have as yet been published; but a writer in the *Indian Medical Gazette*,† in 1881, gives the following quotation from an account of life in Bombay in the seventeenth century :—" After drinking toddy rapidly they took a disease called *barbieri* or *berri-berri*, in which a man tottered in his gait like a dying sheep or span round like a tcetotum." Somewhat similar consequences may of course follow upon alcoholic indulgence without the intervention of Beriberi, but the passage shows that the disease was even then recognised by the Portuguese under the name of Beriberi (which they probably conveyed from Ceylon to the Malabar Coast) as one *sui generis*.

Beriberi is also common enough in Brazil, in the West African negro countries, on the Mozambique Coast, parts of the Indian coast-line, Assam, Indo-China, Sumatra (especially Atjeh), Java, and Malaysia generally as far as Thursday Island. The crew of a schooner, the " George Noble," consisting of ten Europeans and two Chinese, sailed from Sydney to the Gilbert Islands and back in 1889, and during the return voyage were all disabled by what seemed to be Beriberi, excepting three—the master and three others dying. A barque arrived at Onehunga, in New Zealand, a few years before that, from the China seas, with Beriberi epidemic on board; and another, named the " Lothair " is at this moment detained at Nelson, New Zealand, where she put in short-handed for assistance in consequence of Beriberi having broken out with fatal effects amongst her crew and a passenger.

In Australia the disease appears to have been present for several years, though to a great extent unrecognised. It prevailed in the Northern Territory in 1879 or earlier.‡ It was conveyed in a vessel from China to Melbourne in 1888; but had already occurred among the Chinese residing at that port before that.§

Moreover,

* Histoire de l'Isle de Ceylan, Ecrite pur le Capitaine J. Ribeyro, & présentée au Roi de Portugal en 1685.—Amsterdam, M.DCCI.

† Vide *Lancet*, 16/4/81.

‡ Trans. Intercolonial Med. Congress of Australasia : 1889.

§ *Ibid*, 1892.

Moreover, the existence of local endemic Beriberi *foci* among orientals dwelling in Sydney and other parts of Australia has now been established.* And we even hear of an epidemic of it in the persons of Australian aborigines in the Western colony,† and in this case it seemed to have originated *de novo* in a shamefully overcrowded and insanitary lock-up.

Until quite recently Beriberi had only been met with in the United Kingdom in ships arriving from foreign parts where it is endemic: the best-known instance being that of the Chinese crews of two ships of war of that nation sent out in the transport "Too Nan" to join them at Newcastle-on-Tyne, where they had just been built, in 1887.‡

In the Autumn of 1894, however, a distinct and alarming outbreak of this disease occurred in the Richmond Lunatic Asylum, near Dublin,§ where 1,500 patients were confined in an institution designed to accommodate 1,100 only, the condition of which was characterised by the *British Medical Journal* as disgracefully overcrowded and obviously insanitary.‖ This remarkable occurrence has not in so far as I am aware been traced to any specific infection from without, and is consequently the more disquieting to contemplate. Nearly 200 patients were attacked, and the deaths numbered between twenty and thirty.

It is in barracks also in or near Dublin, it may be mentioned, that epidemic cerebro-spinal meningitis, whose ætiological resemblance in certain respects to Beriberi I shall presently point out, has been principally met with in our own country.

So much for the geographical distribution of the disease in so far as it bears upon our situation in this Colony: fortunately it is clear that Beriberi is not yet endemic among the Malayo-Polynesian and Melanesian populations in the Southern Hemisphere.

But it occurs in the Hawaiian Islands, where there is a large Mongolian population; and it was introduced, in 1891, to New Caledonia in the persons of immigrants (or prisoners?) from Tonkin and Annam, and extended there to Melanesian immigrants and New Caledonian natives.¶

It has been conveyed from Asia to San Francisco, and thence by sea to New York;** and was common in London among the Indian and African sailors on board vessels in the docks towards the close of 1892.††

It is in fact a disease which has often been carried by the crews of ships from the East; and one of the many plausible derivations given for its name is *bhur-bahri* (Arab.),—sailors' breathlessness.

7. The Japanese ship of war "Tsukuba" which visited this Colony in 1886, had had a few cases on board during the previous voyage in these seas; but it was just then that an extraordinary reform was being effected in the dieting of the men in

* *Brit. Med. Journal*: 28th September, 1895.
† *Ibid*: 27th October, 1894.
‡ *Lancet*: 23rd July, 1887.
§ *Brit. Med. Journal*: 6th, 13th, 20th, 27th October, 1894.
‖ *Ibid*: 17th November, 1894.
¶ *Archives de Médecine Navale* (Dec. 1891): quoted by Manson, *op. cit.*
** *Brit. Med. Journal*: 29th January, 1887.
†† *Ibid*: 13th October, 1894.

in the Japanese navy, which forms one of the most significant landmarks in the history of Naval Hygiene. The Director-General of the Medical Department of the Navy, Takaki Kanehiro, who received his professional education in London, and is a Fellow of the Royal College of Surgeons of England, having noticed that the food of the ordinary Japanese was very deficient in nitrogenous elements, as compared with the prevalent diets in Europe, succeeded in introducing changes in the diet scales in force in the Japanese navy, where Beriberi was formerly the rule rather than the exception, by which the disease was entirely extirpated from the service in so short a time as three years.

The following tables in connection with this part of the subject were published by Takaki in the Annual Report of the Health of the Imperial Navy for the 20th year of Meiji (A.D. 1887), a copy of which was given to me by Dr. Sasaki of H.I.M.J.S. "Hiyei" when the training squadron of that nation visited Fiji in 1889.

(Extract from Report.)

KAK'KÈ.

This disease, as previously reported, has gradually decreased year by year since the improvement in the scale of diet in the year 1884, and in 1887 it had become entirely extinct.

TABLE SHOWING THE CASES OF KAK'KÈ FOR EACH YEAR SINCE 1878.

Year.	Force.	Cases.	Ratio of cases per 100 of Force.	Admitted to Hospital.	Ratio admitted per 100 cases.	Died.	Ratio of Deaths per 100 cases.	Invalided.	Ratio of Invalided per 100 cases.
1878	4528	1485	32·79	325	21·89	32	2·15	?	?
1879	5081	1978	38·92	485	24·51	57	2·88	8	·40
1880	4956	1725	34·81	319	18·49	27	1·57	9	·52
1881	4641	1163	25·06	300	25·79	30	2·58	16	1·38
1882	4769	1929	40·45	545	28·25	51	2·64	17	·88
1883	5346	1236	23·12	378	30·59	49	3·96	4	·32
1884	5638	718	12·74	209	29·10	8	1·11	1	·14
1885	6918	41	·59	25	60·97	.....	.....	1	2·44
1886	8475	3	·04	.....	.....	.....	.....	.....	.....
1887	9106	.....	.....	.....	.....	.....	.....	.....	.....

From inquiries made I learn that in the more recent period which followed 1887, viz. the four years 1888 to 1891 inclusive, only eight cases altogether of Kak'kè occurred in the Japanese navy, one of them ending fatally, although in 1891 there were more than twelve thousand men on the roll.

8. The old diet-scale contained nitrogenous elements in the proportion of one only to every twenty-eight of carbon. As the normal proportion in the tissues of the body has been found to be about one of nitrogen to fifteen of carbon, Takaki reasoned that nothing less than this would supply the daily tissue waste which the process of life entails. He accordingly obtained the permission of his Government to make some experimental changes in the diets, and prepared a set of twelve alternative tables from which he selected the articles best suited for supplying the need of nitrogen and at the same time adapted for use in a service like the navy. Early in 1884 he introduced a scale of rations in which both elements were greatly increased—but the nitrogenous ones especially, with the result that the ratio of cases of Kak'kè to total strength was at once halved. In that year the proportion was as 1 : 20. Then he added

added still further to the nitrogenous portions so that they stood as 1 : 16, with the result that in 1885, although the force was annually increasing in total numerical strength, the cases of Kak'kè fell to 41 only—equal to ·59 per centum; in 1886 they were only three in number (·04 per centum); and in 1887 and 1888 not a single case occurred. The new ration adopted, by which this magnificent result was brought about, contained rather less in total quantity than those given in 1884–5–6. Its composition is shown in a table (No. 3) which I append at the conclusion of this Minute; and was in the proportion of—

Albuminates	49·70	*momme**
Fats	12·79	„
Carbo-hydrates	185·19	„

Proportion of Nitrogen to Carbon 1 : 16.

This ration is understood to be still in use in the Imperial Navy of Japan.

9. Apart from the enormous gain in human life and health, the value of the labour thus saved to the service by the improved diet must be infinitely greater than the somewhat increased costliness of the new ration itself over the old one. The following table which Takaki has given in the *Sei-I-Kwai* (a medical journal printed in Japanese and English),† serves to throw some light upon the economic aspect of the subject :—

(Extract from "Sei-I-Kwai.")

TABLE SHOWING THE AGGREGATE NUMBER OF DAYS DURING WHICH KAK'KÈ PATIENTS WERE UNDER TREATMENT IN THE DIFFERENT GRADES DURING FOUR YEARS.

Year.	No. of days under treatment at ordinary duty.	No. of days on light work while under treatment.	Duration of rest. Not in Hospital.	Days in Hospital.	Total.
1883	25,235	7,612	7,894	22,534	66,275
1884	10,687	4,675	5,542	11,309	32,213
1885	154	69	175	1,012	1,410
1886	28	.....	71	.....	99

While, then, reading the two foregoing tables together, it seems that in 1883 the navy lost the work of 1,236 officers and men (out of a total strength of 5,346) for an aggregate period of 7,894+22,534 whole days +7,612 half-days—of whom 49 died and 4 were invalided out of the service—the change of diet in 1884 reduced this loss of labour by nearly 44 per cent., and in 1886 the figures became insignificant trifles. Later information asserts that only one death from Kak'kè has occurred in the navy since 1884. Nor was this phenomenal result as regards Kak'kè the only benefit which an improved diet wrought among the Japanese. Takaki reports in the twentieth year of Meiji (A.D. 1887) :—"The number of cases of general "diseases has decreased and kak'kè became entirely extinct; we have consequently "found a decrease in the expenses needed for sanitary purposes. Moreover the body-"weight of the force has greatly increased and its general health has improved "more and more."

10.

* 120 *momme* = 1 lb.

† *Sei-I-Kwai*, May, 1887.

10. The practical lessons to be drawn from these facts should be prominently borne in mind in a Colony such as Fiji, with the interests of a tuber-eating native population to safeguard and the welfare of many thousands of rice-fed Asiatic immigrants to promote. In the first place, however, we may not assume that a deficiency of nitrogen to make good the natural bodily waste is the sole or active cause of the condition we call Beriberi. There is in fact fairly conclusive evidence to the contrary: and the opinions of such medical men as have had experience of the disease in its proper habitats are pretty well agreed that the diet question is one which mainly determines only the susceptibility of persons to invasion by a specific poison. That poison, as might be expected, has been made to reveal its bacterium, its micrococcus also, and even mycelial growths in the blood. Manson regards some of these discoveries rather sceptically, and—himself the soundest and most eminent of English tropical pathologists—admits that he has always failed to find a distinctive micro-organism of Beriberi.*

Dr. de Lacerda, however, at Rio de Janeiro, claims† to have first isolated the bacterium of Beriberi in 1883, and to have produced, in every instance, a train of symptoms similar to those exhibited by the disease in man when he inoculated rabbits and monkeys with the microbe, both in the fresh state and after several successive cultivations. All Lacerda's inoculation experiments terminated in the death of the animals, and these effects followed even on inoculation performed with bacteria captured in the atmosphere of infected localities.

11. The gravity of the disease from a State Hygiene point of view is not diminished by the fact that in India and in Brazil, at least, a similar affection has been noticed in horses and swine,‡ and it has been abundantly asserted that the bacteria of Beriberi are capable of infecting several other animals and birds, among which are rabbits,§‖¶**†† hares,¶ monkeys,§¶** dogs,‖** guinea-pigs,§†† rats,** and pigeons;** and that in air, soil, water and food, as well as in the human blood, the micro-organisms have been found and can by direct inoculation, or after culture, reproduce the symptoms of Beriberi in such animals.

12. But it is some consolation to know that the best authorities‡‡ agree in believing that residence of, at the very least, some weeks' duration in an infected place is an essential factor in producing Beriberi in a person who has not previously suffered from it; and that even a victim may rid himself of the infection, if his case be not too far advanced, by removing from the infected district to one where Beriberi is not endemic, nor, of course, for the time being, epidemic,—provided the climate be a cool and dry one.

In order to succeed in this, however, mere removal is not enough. In addition, attention must be paid to all the more important dictates of hygienic science

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* Op. cit., p. 481.

† *Lancet*, 27th November, 1886.

‡ Peste de Cadeiras, ou Epizootia de Marajó; mas analogias com o Beriberi,—J. B. de Lacerda.—Rio, 1885.

§ De Lacerda: *Lancet*, 27th November, 1886.

‖ Pekelharing: *Lancet*, 24th September, 1887.

¶ Ogata: *Lancet*, 30th July, 1887.

** Wallace Taylor: *Sei-I-Kwai*, August, 1885.

†† Musso and Morelli, of Monte Video: *Gaz. Méd. de Paris*, 21st January, 1893.

‡‡ Pekelharing and Winkler: Op. cit.

science. Foremost, the diet must be regulated so as to yield the necessary proportion between the nitrogenous and carbonic constituents (which may, as shown by Takaki, be estimated at 1 : 15 or 16), and to afford a sufficiency of them both.

In the second place, abundance of pure air must be ensured to the patient by choosing a dry, non-malarious, elevated, and airy site, and a well ventilated, sparsely tenanted, and well drained building as his dwelling-place.

Thirdly it has been noticed, as pointed out and corroborated in Dr. Joynt's Report, that the influence of humidity and heat are potent factors in determining what Manson aptly terms the 'explosion of the disease,' in persons predisposed to it who have been brought into relation with its specific poison. To avoid or suppress it the temperature must never exceed that at which fires indoors can be dispensed with in comfort,—about 60° Fahr.

Lastly, attention must be scrupulously paid to the state of the soil—which Pekelharing and Winkler go so far as to recommend should be sprayed with a solution of corrosive sublimate.

13. If we compare these measures with the circumstances by which the introduction of the unfortunate Japanese coolies to Fiji in 1894 for the Colonial Sugar Refining Company was attended, we find that, assuming one or more of them to have acquired the poison in their own country and that it was latent in them when they arrived here, they then met with several of the conditions most essential for 'determining the explosion' of an epidemic of Beriberi.

Their diet, as quoted in the accompanying Reports, was inferior in nitrogenous material to the proportion insisted on by Takaki.

The sites selected by the Company on which to house the immigrants were low-lying, flat, alluvial, damp, and the Nausori one was naturally ill-drained. The buildings themselves were peculiarly designed, to meet the requirements of the Japanese emigration authorities, their ventilation being marred by reticulations and numerous partition-walls—some of iron and some of rough unplaned wood, and iron roofs, unbearably hot in the daytime and, by contrast, chilly in the small hours of the morning; but well adapted for retaining disease germs.

Their work was hard, in low-lying, miasmatic, alluvial canefields. At Wailevu they adjoined a wide belt of mangrove-swamp, and the men were much exposed to a hot sun, while at Nausori the land overlies extensive beds of river-clay and old mangrove-swamps, and rainy weather was frequent and prolonged.

At both places they met with great heat and a moist atmosphere, to which they were not accustomed; and at times they were most injudiciously set, although new arrivals, to clear new land and dig drains in sour swampy soil: and, as to the soil surrounding their dwellings, it may be stated that in an adjoining building at Nausori, used as a barrack for European artificers about the mill, dysentery, hepatic abscess, and enteric fever occurred, and ended fatally in several instances.

14. Though we enjoy an immunity in this Colony from malarial diseases in the common acceptation of the term, it has been repeatedly shown that Indian coolies and other persons who have suffered from malarial poisoning before coming

to

to Fiji, are apt to be attacked by remittent and even intermittent fever here. Such attacks have been especially common among the Indian coolies at Labasa, who, in 1894, were fellow-workers with the Japanese immigrants, and have been clearly defined by the District Medical Officer, Dr. Noble Joynt, who had a specially large experience of zymotic and other febrile diseases before he was appointed to this Colony, and is in all respects a careful and highly competent observer.

Beriberi was at one time thought to be a form of malarial cachexia. It is still, even in the light of modern bacteriological research, pretty well agreed among writers on Beriberi, that malarious surroundings constitute one of the causes which predispose to its occurrence; and the two conditions have, in fact, many affinities in common. The local factors which cause malarial saturation in the Indians to recrudesce in Fiji may be therefore assumed to assist in predisposing physiologically susceptible persons to be attacked by Beriberi, when brought into contact with its specific infection.

15. The only other diseases *usually quoted* as liable to be confused with Beriberi are scurvy, pernicious anæmia, and ankylostomiasis, and the sleeping sickness or negro lethargy of the West African peoples.

16. A diet which will cause scurvy may be thought to predispose to such a disease as Beriberi, and, as already mentioned, Gemelli relates how both existed concurrently on board the Manila galleon in which he was a passenger. Modern observers do not, however, trace any connection between the two, and believe that the well-to-do and fleshy may fall victims to Beriberi as readily as any other people. It seems more probable that the scorbutic state has been merely confused with Beriberi, on account of the rough similarity between some of their external symptoms, and the parallelism of their prophylaxis in respect to food.

17. As regards ankylostomiasis the case is not so plain. But the distinction is one of great importance to us in this Colony; inasmuch as the Indian coolies here have shown themselves to be especially prone to become the hosts of the worm, invasion by which constitutes this last-named disease, if it be one.

The presence of the *ankylostoma duodenale*, a small nematode worm which likes to live in that portion of the human intestine which leads onwards from the stomach, and in appropriate subjects may infest it in hundreds or even thousands, is often associated with the condition of anæmia, into which so many of our coolies drift. A report on Anæmia or the Beriberi of Ceylon which was drawn up by the Principal Civil Medical Officer of that Colony in 1887, treats of the ankylostoma; and Dr. Kynsey confidently supports Erni's view that this worm is responsible for the production of anæmia, and that the disease which results is one and the same with Beriberi. This opinion was a tempting one, as the obvious remedy was merely to extirpate the intruder (though this is no easy task) and to exhibit iron, with good food. Its publication led to the employment of the terms—Ceylon Beriberi and True Beriberi; the latter being reserved to denote the specific peripheral multiplex neuritis which forms the subject of this Minute, in contradistinction to the cases of anæmic lethargy known also as Beriberi in Ceylon, and often associated with the presence of entozoa.

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* Drs. Pekelharing and Winkler.

18. Since the investigations and deductions of the Dutch Commission* in Sumatra, however, it has become the generally accepted view that Beriberi is Beriberi: and that while, undoubtedly, persons suffering from that disease are often anæmic, and may at the same time harbour large numbers of ankylostomata in their intestines, still, these conditions are essentially different from one another in their causation and pathological nature.

19. Anæmia has in most countries, and for many years, been looked upon as a part and parcel of the features of Beriberi. The District Medical Officer for Rewa shared that view so lately as in 1894; but his statement then would seem to have been based on the published opinions of other observers in countries he had not himself visited.* Wallace Taylor, however, who made extensive examinations of the blood of Kak'kè patients in Japan, disclaims for Beriberi a direct relation to the accompanying anæmia, if any, and states that the severity of the disease cannot be gauged by the degree of anæmia present.† The attached Report by the District Medical Officer for Rewa, shows that he has now arrived at the same conclusion by similar means in the case of the immigrants here; and this opinion has the merit of being founded on actual experience. In the matter of anæmia his cases differed materially from those of Dr. Joynt, at Wailevu; and the contrast is somewhat unexpected.

20. As regards the presence or absence of ankylostomata, there is something to be said. There are two methods of determining this question; one applicable during life and the other by post-mortem examination of the gut. The existence of this entozoon in Fiji, was first noted by my predecessor, Sir William McGregor, in 1876, in the intestine of a native, and again in 1879 in two instances, but without pathological complications; but it was not until 1888 that it became generally known or that its prevalence among the Indian immigrants was regularly observed. In a report (C.S.O. $\frac{1279}{1888}$), which I prepared in that year and submitted to His Excellency, I drew attention to the increasing prevalence of a progressive form of anæmia among the Indian coolies on alluvial sugar-plantations in the Colony, and I referred to the case of Gopal (reg. no. 4042), in whom, associated with marked anæmia, fixed epigastric pain, hæmic murmur, præcordial oppression, breathlessness, general œdema, numbness of the shins, paresis, prostration, and somnolence, I found, *post mortem*, about 1,100 specimens of the *ankylostoma duodenale* in the small intestine, on the 5th of April in that year. I pointed out the probability of ankylostomiasis being met with amongst the coolies to a serious extent and perhaps in circumscribed epidemics,—occurrences which have since been realised; and I indicated the desirability of an investigation being made in the localities where coolies were employed, with a view of taking precautionary measures against this pest becoming general.

I received Dr. Kynsey's Report‡ on almost the very day when I examined Gopal, and in my communication above referred to, I stated that "Whatever may "be the safety of assuming the coolie anæmia in this Colony to be identical with "Beriberi,

* Pernicious Anæmia—Report on; by Charles T. W. Hirsch, District Medical Officer.—Suva, 1894.

† *Sei-I-Kwai*, May, 1886: quoted in the *Practitioner*, August, 1886.

‡ Anæmia, or the Beriberi of Ceylon,—W. Kynsey, P.C.M.O.—Colombo, 1887.

" Beriberi, it is now plain that it may be associated with anchylostomiasis; but it " yet remains to demonstrate their relation to one another as effect and cause."

Prior to that I had recorded a series of cases which were admitted to the Colonial Hospital here for anæmia, as being in reality cases of Beriberi. They presented all the symptoms quoted as belonging to Gopal except the worms; but I founded my diagnosis chiefly on the description of Beriberi given by the late Surgeon-Major Horton (himself a native of West Africa), whose statements* are admittedly now out of date, and do not exhibit that definition which the observations and discoveries of the last twenty years have produced.

21. It is nevertheless proper to state that the District Medical Officer for Taviuni, Dr. Finucane, who has had tropical experience in the Gambia, finds a number of cases among the Indian coolies at the Holmhurst plantation, with the same train of symptoms, in whom the signs of peripheral neuritis are unequivocal; and in the survivors of whom an increase in the nitrogenous elements of their diet has caused a steady increase of body-weight, and an encouraging improvement in their general condition.† He believes these to be genuine cases of Beriberi, and this opinion seems well founded; though it must be accepted with the reservation, of course, that the degree in which two or all three of these diseases may concur, varies with the individual. Dr. Sonsino, of Pisa, a physician of wide experience in all that relates to parasitic diseases, asserts Beriberi to be a polyneuritis unconnected with the ankylostoma or any other intestinal worm, pointing out the significant fact that while both diseases are commonly met with in Ceylon and some other localities in one and the same patient, yet in Egypt and in northern Italy, where the *ankylostoma duodenale* is of notably frequent occurrence, and its effects are well known, Beriberi is never met with.‡ Manson, in his monograph,§ makes the same pertinent remark.

22. This Colony has during the late epidemic afforded a useful field for observing the relation, had there been any, between Beriberi and ankylostomiasis; inasmuch as in both localities the Japanese, reeking with the Beriberi poison, and the Indians, common victims of ankylostomatic anæmia, were employed by the same proprietors on the same plantations, and were moreover in daily association with the Fijian natives who dwell in the adjoining villages. Yet the Japanese did not suffer from ankylostomiasis; no epidemic of Beriberi—no sporadic cases even—broke out among the Indians; and the Fijians—pathologically satisfied with their own familiar *filaria sanguinis*—resisted both the foreigners' diseases successfully.

23. Our Fijian experience therefore goes to further establish the lately prevailing belief that Beriberi is a disease *sui generis*, and is not the effect of *ankylostoma duodenale*, and is not necessarily associated with it or with anæmia.

24. At any rate it is now certain, from our experience in Fiji as well as from that gained in other countries, that profound or even fatal anæmia may attack persons and yet no more than half-a-dozen ankylostomata be found in their intestine *post mortem*—that (true) Beriberi may attack persons without either ankylostomiasis or anæmia coexisting with it—and that a few ankylostomata, certainly thirty

* The Diseases of Tropical Climates and their Treatment; by J. A. B. Horton, M.D.—London, 1874.

† Medical Reports on the District of Taviuni for February, July, and December, 1895.

‡ *Lancet*, 22nd February, 1890.

§ Op. cit.

thirty and perhaps hundred, may be present in the intestine without causing either of these diseases.

25. There are important lessons to be learnt from these contrasts in connection with the means to be used for delimiting the spread of Beriberi. The removal of the surviving Japanese to their own country was effected in February last; their houses were washed and disinfected with corrosive sublimate as described in the Reports; and, subsequently, those at Labasa were utilised for the reception of Indian coolies.

Eleven months have now passed over and the moist hot season has again come round, but no new cases have been recognised. We may therefore expect that the disease was stamped out by the measures taken, although it is the general belief that persons may inhabit a Beriberi centre and not acquire it until many months' or even two years' residence there. Still, the complete removal of the Japanese immigrants, and the disinfection of their dwellings (including the subsequent demolition of the one at Nausori), should be enough to preclude our regarding the sites any longer as Beriberi centres.

26. It is unnecessary to allude to the African sleeping sickness here, as nothing has been met with out of Africa which quite corresponds to it.

27. But there is just one other point upon which a note should be recorded; and that is, the parity between Beriberi and epidemic cerebro-spinal meningitis, a disease of which we had a sufficiently alarming example in this Colony in 1885. They may be said to run in parallel but widely-separated grooves. Beriberi is a peripheral nerve affection in which invasion by specific bacteria causes atrophic and degenerative changes in the nerve tissue; while the other is a lesion of the central nervous system, in which a similar cause tends to the production of inflammatory and even suppurative results chiefly about the sheaths which invest the brain and cord substance.

Yet, saving as to the influence of atmospheric temperature, in regard to which one is the converse of the other, the general behaviour of the one during its epidemic prevalence strongly resembles that of the other; and in their sporadic examples they are not dissimilar. But this is perhaps not very remarkable, the same great anatomical division of the body being primarily involved in the two diseases.

28 The death-rate in epidemics of Beriberi in other countries has varied, according to the published reports, between very wide limits. I have therefore merely recorded in a table (No. 2) the number and ratio of deaths which occurred in the places to which the accompanying Reports relate, without further comment.

29. In conclusion I append a tabular resumé of the two Reports herewith submitted; and also a copy of Takaki's improved ration as adopted for general use in the Japanese navy which, it will be noticed, is considerably more nutritious than that stipulated for by the Japanese emigration authorities with the Colonial Sugar Refining Company.

B. GLANVILL CORNEY,
14/1/96.

No. 2.

No. 2.

Table Comparing and Contrasting the leading Characteristics of the Cases at (1) Nausori, and (2) Wailevu.

		Symptoms, &c.	Nausori.	Wailevu.
Statistical.	.....	Total number of Japanese	50	250
	.....	Sexes and ages	All males from 18—30.	All males from 18—28.
	... ..	Number of cases of Beriberi	42	226
	.....	Ratio of cases to total number of Japanese	84 per cent.	90·40 per cent.
	.....	Deaths	8	57
	.....	Mortality per centum of cases.....	19·04 per cent.	25·22 per cent.
Nervous System.	Mental	Delirium, drowsiness, torpor	* * *	None.
	Sensory	Degree of prostration at first	Disinclination for work.	Weariness in the legs.
		Pains and tenderness	In calves and other muscles.	Especially in calves.
		Anæsthesia	Of shins in all cases. Later of thighs, insteps, wrists, hands, forearms, shoulder. Then of abdomen.	First of calves, shins, and feet; later of thighs, wrists, and fingers. Lastly, of trunk.
		Sensibility to heat, cold, and electric stimuli	Concurrently diminished.	* * *
		Hyperæsthesia	* * *	Occasionally marked.
	Motor ...	Loss of power	First in peronei, abductors, and flexors of knee; then supinatores longi and extensors of hand and fingers. 'Wrist-drop' in fatal cases. Laryngeal paralysis with aphonia in 4 cases.	First in muscles of calves, shins, and insteps; later in hands, fingers, and forearms. 'Drop-wrist.' Paralysis of abdominal and, lastly, intercostal muscles. Complete paraplegia in 31 cases. Marked ataxia of gait in 17 cases.
		Subsultus	Diaphragm and abdominal muscles in 6. Twitching in 2. Hiccough.	* * *
		Cramps	Often.	Sometimes.
	Reflex ...	Reflexes—		
		Patellar...	Absent in all.	Lost in 37 cases; good in several; exaggerated in some.
		Abdominal	Usually preserved, but disappeared before death.	Lost in 12 cases; unilateral in 1; weak in 2.
		Cremasteric	Do.	Lost in 6 cases; unilateral in 2; weak in 2.
		Epigastric	Do.	* * *
		Ankle clonus	Never noticed.	Never elicited.
Circulatory.	.....	Anæmia	None.	Nearly always.
	.....	Œdema	Over crests of tibia all cases. General in 4. Hydrothorax 4 cases. Ascites 2.	General in the 'Wet' cases. Of penis 4; of scrotum 2. Hydrothorax 4. Ascites 4. Pericarditis always met with *p. m.*
	.....	Palpitation	Common	Usual.
	.....	Heart-sounds	Occasional systolic bruits in 9 cases.	Lengthening and roughness of 1st, and reduplication of 2nd, invariable. Hæmic systolic murmurs occasionally. Apex beat diffused and often tumultuous.
	.....	Area of cardiac dulness	Extended in 5 cases.	Extended.
	.....	Pulse	Weak and easily accelerated by exertion. Dicrotic and irregular in all severe cases.	Nothing characteristic; but easily accelerated on slight exertion.
Respiratory	.....	Breathlessness	Occasional at first: marked in all later on.	Common, with pain across chest.
	.....	Œdema of lungs	10 cases.	* * *
Digestive.	.....	Tongue	* * *	Pale and slightly furred usually. Bright red with enlarged papillæ in many.
	.....	Appetite	Unimpaired.	Anorexia frequent.
	.....	Dyspepsia	Indigestion and flatulence common and distressing. Epigastric pain.	Flatulent dyspepsia, frequent. Epigastric pain, frequent.
	.....	Emesis.....	Not unrare: obstinate and serious in 6 cases preceding death.	Often ushered in symptoms. Severe in 18 cases towards end.

		Symptoms, &c.	Nausori.	Wailevu.
Digestive.	.....	Bowels	Constipation more common than looseness.	* * *
	.....	Hepatic	* * *	Liver sometimes enlarged; several nutmeg. Slight jaundice, 16 cases.
	.....	Anchylostomata	A few in 3 cases.	* * *
Urinary.	.....	Urine	Usually normal. Quantity greatly diminished in the 'Wet' cases.	Albumen: never. Retention: 3 cases. Dysuria: 4 cases.
Integumentary.	.....	Skin eruptions, &c.	None.	Carbuncle, 1 case. Boils, 2 cases. Skin harsh, dry, lustreless, and scaly in atrophic cases; soft and often shining in œdematous ones.
	.....	Anæsthesia	In parts, in all cases.	Common in parts.
		Pyrexia	None while under treatment for Beriberi. Subnormal temp. preceding death in some cases.	Occasional and slight in about 30 cases.
		Complications	Prior to Beriberi, diarrhœa in 30 cases, fever in 10.	Dysentery and dysenteric diarrhœa, 32 cases; remittent fever, 20 cases.

No. 3.

(Vide paragraph 8.)

AVERAGE DAILY QUANTITIES OF EACH ARTICLE IN THE RATION FROM 1884 TO 1887.*

Article.	Average daily amount for one person.			
	1884.	1885.	1886.	1887.
Rice	249·99	153·45	120·06	78·39
Barley	.....	60·55	78·76	43·65
Bread	2·56	37·11	53·15	108·72
Meat	53·39	59·00	58·68	64·14
Fish	53·28	30·57	43·81	24·57
Vegetables	104·49	127·66	136·92	123·45
Miso	16·30	16·94	19·27	9·37
Shoyu	24·37	26·46	24·99	21·12
Eggs	23·87	19·40	16·15	10·50
Beans	·39	·92	1·10	·42
Sugar	4·13	9·64	11·35	9·72
Fat	·56	·73	·63	2·52
Tea	2·02	1·65	2·14	·90
Salt	·94	1·05	·87	1·29
Pickles	60·97	62·90	58·29	31·95
Vinegar	1·40	·95	·54	·36
Alcoholic liquors	3·92	6·70	5·37	2·70
Fruit	3·07	1·60	1·26	·66
Milk	1·88	8·06	11·23	28·95
Spices	·13	·13	·05	·18
Total	607·66	625·53	644·62	563·58

Weights in "Momme."

(1 *momme*=about 58 grains Troy. 120 *momme*=about 1 lb. Av.)

* From "The Annual Report of the Health of the Imperial Navy" (of Japan), 1887.

No. 4.

No. 4.

(Vide paragraph 8.)

WEIGHT OF NUTRITIVE ELEMENTS *per* MAN *per diem*, SHOWING PROPORTION OF N AND C.*

Year.	Albuminates.	Fats.	Carbo-Hydrates.	Proportion of Nitrogen and Carbon.	
				N.	C.
1884	52·17	11·67	206·16	1	16
1885	52·43	12·13	211·95	1	17
1886	56·73	12·86	204·66	1	15
1887	49·70	12·79	185·19	1	16

Weights in "Momme."

* From "The Annual Report of the Health of the Imperial Navy" (of Japan), 1887.

No. 5.

No. 5.

Report on an Epidemic of Beriberi that occurred during 1894 in the Rewa District.

District Medical Officer, Charles T. W. Hirsch, M.R.C.S. Eng., L.R.C.P. Lond., F C.S., &c., &c., to The Honourable the Chief Medical Officer.

Sir

I have the honour of reporting to you the following facts concerning the recent epidemic of Beriberi, which occurred among the Japanese labourers residing and working on Nausori Sugar Plantation in the Rewa district of Fiji.

This estate is situated about 10 miles from the mouth of the Rewa River, and consists of alluvial flats of, in all, some 1,500 acres, which extend from the river-bank back to, and in between, the hills, of which there are many.

The following table gives the meteorological observations as taken at Nausori Plantation, and for permission to publish which I have to express my thanks to the Manager in Fiji of the Colonial Sugar Refining Company.

Month, 1894.	Temperature.		Rainfall.
	Max.	Min.	
January	85·6	69	21·51
February	86·5	70·78	8·75
March	85·8	70·76	7·38
April	83	69	10·51
May	81	67	11·96
June	79	65	4·96
July	79·4	65·4	4·45
August	80·1	66·3	7·81
September	83	67·9	9·88
October	81·6	67·9	12·01
November	82·2	68·2	19·38
December	85	70·6	12·58
		Total Rainfall, 1894...	131·18 inches

History.

The Japanese arrived in Fiji on 27th April, 1894, having come direct, per s.s. "Afghan."

I understand that they had all been medically examined in Japan and passed as healthy and fit for agricultural labour. No medical diary was handed in by the ship's surgeon (a Japanese). I was informed that diarrhœa and *mal de mer* were the only complaints that came under treatment during the voyage.

They were all of the male sex, and their ages varied from eighteen to thirty. They belonged to the class of farm-labourers, and nearly all came from the interior of Japan. Some confessed that they came from districts in which Kak'kè was endemic, but they all declared that they never had the disease.

They came under agreement to work on sugar-plantations belonging to the Colonial Sugar Refining Company.

Under

Xmas. 1st Hospital diets

1 Sack Wheatflour every 3 or 4 days

March 1st Sunday

White beans	22 katis		Say 6.5 oz	
Broken rice	8 "	72 patients	2.2 oz	Grams not 2
Dried Greenpeas	5 "		1.5 oz	
Brown Sugar	7 "		4.1 oz	
White Sugar	7 "			
Pork	15		4.4 oz	

Say 1/4 bag of wheatflour

2nd

White beans	38 katis	66 pts.	11 oz	@ 2% 0.22 fat 270 gms N
B. rice	15 "		4.6	@ 0.9 0.03% fat
W. Sugar	8 "		2.6	
Wheatflour				

3rd

White beans	32 "	64	
B. rice	15 "		
Sugar	8 "		
Wheatflour			

4th

Rice	48	64	16 oz	20.6 @ 0.9 about 0.165
B. Rice	14		4.6	
W. Sugar	8		2.6	
Chinese beans	8		2.6	
Pork	13		4.3	at 73% 3.1 oz fat
Wheatflour.				

BB outbreak began 5th MCH Beans ran out on 3rd Onions stopped 21/XI/02
Reckoned on pork days each man only gets about 2 taels

Last dried fish on 7th ? Feb

Beans & some rice off ship on 11th
but much not off till 21/3.

11/3 Evening meal. Bean meal. Bean pudding. Dried oysters. Rice

about 100 coolies at Kagzi dining hall tiffin

Coolies. 17/3 Salt turnips. Pickled onions. Rice } Evening meal

19/3 1/4 tub vermicelli boiled & few oysters. midday.

Christmas Island

? 3/1903

	Average consumption per month for 6½ months VIII 02 – II 03	Estimate 500 men daily consumption per man	Estimate for 500 men per month.	
Rice	98.4 bags	25.5 oz.	@ 28 oz per day every day = N = 98.00 C = 4426.2	253 pikuls
Pigs	24.4 at 80 katis less 5% for bones &c	7.4 taels	@ 4 taels twice a week. 8 taels " "	about 13 pikuls 26 pikuls
Peas & Beans.	5.5 bags = 11 pikuls		2 oz. 5 times a week = N 30.8 = C 294.4	8½ pikuls
Vermicelli	4.4 boxes = 11 pikuls	1.94 oz	2 oz 5 times a week	8½ pikuls
Salt Fish	2.4 tubs = 9.6 pikuls = 1279.68 lbs.	1.89 oz	4 taels 5 days a week. = N 233.7 grs = C 157.08 grs	25 pikuls
Dried Fish	1 bundle = 126 lbs.			
Pres'd Salt vegetables	101.0 jars			
Pres'd Dried vegetables	11.2 boxes, baskets etc.			
Sugar	6.7 baskets/boxes = 16.75 pikuls	2.3 oz.		
Lard	2.1 kegs		2 oz. 5 times a week.	8½ pikuls
Onions	2.4 bags		½ oz twice a week.	84 katis
Sweet Potatoes	7.2 bags		8 oz twice a week.	13½ pikuls

28 oz Rice
2 oz Peas
4 Taels D. fish
} C = 4877
N = 362.5

2 oz Lard

		N	C
Rice	24 oz	88·8 grains	3893·92
Peas	1 oz	15·4	147·2
D° Fish	4 Tabls	233·7	157·08
		337·9	4198·2
Land.	2 oz.	—	672
		337·9	4870·2
Maccaroni	1 oz.	6·3	169

P_2O_5 -

Rice 25 oz.		0·298 grs
Peas 1 oz	~~6·8288~~ grains	~~40~~ ~~250~~
D fish 5⅓ oz	~~9·624~~	9·624
		49·922 grs P_2O_5.

TR Lewis Indian gaol. veget

Grain	28 oz (including 4 oz pulse)
Greens	6
Fatty	¼
Salt	½
Condiments	¼

Stock in sight
11/3
roughly

~~350~~ 239 bags

22 pigs

13 bags

8 boxes

2 tubs
1 bundle

not very much
3

7 boxes?

5? tubs

4 bags

—

Some Pumpkins & yams.

The following while not a perfect diet would be a considerable improvement on the present scale.

Rice	26 – 28 oz	daily
Dried or Salt fish	4 · tuels	5 days a week.
Pork	not less than 4 tuels	2 days a week
Lard	2 oz	on fish days
Sugar		
Vegetables:—		
fresh: yams, sweet potatoes, pumpkins, greens stuff		4–8 ozs.

onions added to but not replaced by dried or tinned preserved vegetables.

Peas, Beans, Barley, Dhal, Vermicelli etc	1 or 2 ozs. daily

Under their contract they were to receive such daily rations as are named on Enclosure marked D, lodging, medical care, and one shilling for each day's work they performed. This arrangement entailed, I understand, work per day, and not by the task.

Fifty of them came into this district.

In July there was, unfortunately, some friction between their employers and themselves, and it was then that I first saw them professionally—many of them claiming exemption from work on the grounds of ill-health.

Seventeen stated that they had Kak'kè, and the majority of the remainder that they felt disinclined for work.

In only two cases could I then detect even slight anæsthesia of the knees; and I must confess the diagnosis of malingering crossed my mind: and if it had not been that I had recently read Dr. Cantlie's translation of Pekelharing and Winkler's treatise on Beriberi, I should most surely have entered 'Shamming siek' against their names in the Hospital Case-book.

In August, I came to the conclusion that the disease they were suffering from was, as they themselves stated, Kak'kè or Beriberi.

General Facts.

In all forty-two cases came under observation. Of these—eight died, thirty-four were returned home suffering from Beriberi, while the remainder of the fifty who came here were likewise sent with the rest to their country in February, 1895. The method of inquiry pursued, was to keep a history of each individual case, and especially to investigate the areas of cutaneous anæsthesia, the electrical reactions of the muscles, and to make microscopic examinations of the blood with a view to the detection of such bacteria as were found by Musso and Morelli, and Pekelharing and Winkler. The stools were likewise examined for anchylostomata, and general notes of the progress of the cases were kept.

To save space and, what would have been in many instances, useless repetition, I have only included with this separate histories of two individual cases, but I have given in the text such general facts as appeared to me worthy of consideration.

Previous History and Habits.

As stated before, the Japanese were, according to the ship's surgeon, healthy on arrival in this Colony.

At Nausori, they lived in a house specially built for them on the flat ground near the mill. The dwelling had a galvanised-iron roof, and was divided by partitions of the same material into cabins.

The ventilation was good, and the cubic space provided for each man was about 400 cubic feet, that is 100 cubic feet more than the Indian Immigration Ordinance of 1891 specifies should be allowed for each coolie. A sufficiency of tanks adjoined the house and collected the rain-water from the roof. Arrangements were made so that they could each indulge in a warm-bath every evening.

The

The work the men were given to do was chiefly that of cane-cutting, which is certainly heavy work in such a hot and moist atmosphere as that of Fiji.

As Beriberi is practicably endemic throughout Japan, it is natural that many of the immigrants who were sent here came from districts in which that disease prevails, but they all assured me that they had never personally suffered from it.

Prodromous Symptoms.

These certainly seemed to be vague and ill-defined. A considerable number of the patients suffered from diarrhœa on the voyage; and out of the forty-two cases treated, some thirty had, previous to the diagnosis of Beriberi, been under treatment (in Fiji) for diarrhœa.

One of the earliest symptoms found, is what, I fear, occurs also in people who do not develop Beriberi, viz.—a disinclination for work. All the Japanese complained of this, long before I was able to form any positive diagnosis. Ill-defined tenderness about the calf of the legs and occasional shortness of breath, should also be mentioned as some of the first indications of this complaint.

Ten cases had previously, in Fiji, been in hospital for pyrexia.

General Incidence and Progress of Symptoms.

These invariably seem to gradually supervene on the prodromic stage. One day a patient might complain of loss of sensation about both knees, which would appear to be slightly œdematous, gradually the numbness would extend, and at the same time the patient might complain of cramp, and state that the muscles of the calf felt stiff, later on, a certain amount of paresis would show itself, and the gait become characteristic and develop into a shuffle.

In some cases, and these constituted the majority, the anæsthesia would extend to the thighs, arms, and even to the hands; and sensation would be lost—not only to touch and pain, but also to heat, cold, and electric stimuli. In a few cases (four) the œdema became excessive and spread to the abdomen and face; but in the greatest number, the initial puffiness observed about the knees did not extend. In all, especially the severe cases, shortness of breath was a marked symptom.

In severe cases the laryngeal muscles may be affected and aphonia result. This was well marked in four of the cases.

General Analysis of Symptoms.

Enclosures B and C give brief histories of two typical cases,—one in which death resulted, while in the other there was a tendency to improvement, whether such was permanent or not the removal of the Japanese from this Colony prevents me from saying.

The Integuments.

As there was no anæmia in any of the cases, consequently in none of them could any alteration be noticed in the integuments, except where the œdema caused the skin to be stretched and thus appear pasty. Neither was there any eruption. The œdema, like the anæsthesia, started in all the cases symmetrically over the crests

crests of the tibia. In thirty-eight cases it did not extend beyond them, though the ankles were occasionally slightly puffy.

In four cases the Beriberi was of the kind known as 'wet'; and in these the œdema spread rapidly, and general anasarca supervened. In two, ascites caused such distress that paracentesis abdominalis had to be resorted to.

Neuro-muscular System.

Anæsthesia of the skin.—This, the first symptom of Beriberi, always commenced with the œdema over the front of the leg. In eight cases, it did not extend beyond this part; in thirty-four it did.

Its method of progression seemed to be uniformly the same: first upwards, along the inner side of the thigh and downwards to the dorsum of the feet. Subsequently, as was observed in eighteen cases here, the arms were also involved, commencing with the inner surface of the wrist, and passing from there to the back of the hand, and thus along the inner side of the arm to the shoulder. In thirty-four patients, the lower part of the abdominal wall was anæsthesic. I could never detect the numbness about the mouth, which is said to be so general by Simmons.

Sensibility to heat, cold, and electricity, diminished equally with that of common sensation.

In no case could any history of preceding hyperæthesia of the skin be obtained. The muscles, especially those of the calf, were frequently excessively tender.

Cramp was often complained of.

Epigastric pain, especially after a meal, was frequently most marked; the patients describing it as being most severe in character; and to use their own words, "like a hot ball rolling about in their stomachs."

Reflexes.—In all the cases from the very first, I was unable to obtain any patellar reflex.

Ankle clonus was never noticed.

The skin reflexes, that is—cremasteric, abdominal, and epigastric—were usually preserved; they, however, disappeared in the fatal cases, shortly before death. The pupil reflex was always normal both to light and accommodation.

The Muscles invariably showed diminished irritability to both the galvanic and faradic currents.

In eighteen cases the reaction of degeneration was present.

It is especially interesting to note that every Japanese that came to Rewa, including the Inspector, showed, for the muscles supplied by the peroneal nerve, an altered electrical reaction; which, though, perhaps, could not be classed in all cases as the reaction of degeneration: still the response to electrical stimulation was more marked for the galvanic than for the faradic current, and in many cases the anodal closing contraction was stronger than the cathodal closure contraction.

The order in which the various groups of muscles were affected was always the same; those supplied by the peroneal nerve being first implicated, and later on the

the abductors and flexors of the knee, and then the supinator longus and the extensors of the hands and fingers. Wrist-drop was noted in all the fatal cases.

In four cases the laryngeal muscles were involved.

In six of those who died the diaphragm and abdominal muscles were affected, and this seems to be one of the chief causes of death.

In three cases I managed to examine microscopically portions of the vagus and phrenic nerves, and these all showed signs of parenchymatous change.

The Circulation.—In all the cases the pulse was weak and became accelerated by exertion. Dicrotism and irregularity were noticeable in all the severe cases.

Palpitation was a common symptom.

In nine patients occasional systolic bruits could be heard; in but five did the area of cardiac dulness seem to be markedly increased, and in three of these it was on account of hydropericardium.

The Blood.—In absolutely none of the cases that came under observation could any anæmia be detected: in fact all the Japanese seemed to be full-blooded, robust, peasants. The blood was examined in some ten cases; and in all these the number of corpuscles and the amount of hæmoglobin was normal; in two cases it was above the normal. In none were the number of white corpuscles in excess, nor were any poikilocytes to be noticed. Numerous preparations of blood were made with a view to the detection of germs. In making these, care was always taken to wash the finger of the patient with soap and water, and afterwards with ether before prieking it to obtain a specimen.

The first drop of blood which exudes was wiped away and then the finger was pinched, and as soon as a drop not larger than a pin's head appeared, the same was taken up by lightly touching it with the centre of a cover-glass. Fuchsin and methylene blue stains were used. Search was then made with a one-eighth objective. On many occasions nothing abnormal could be seen.

In nine instances some very small rod-shaped bodies were discovered. When such were noted, attempts were made to obtain cultivations by dropping a little blood, both on to Liebig's extract and on to gelatine kept in sealed tubes.

Unfortunately in no instance could I obtain any result.

The Lungs.—In the four cases of wet Beriberi there was a certain amount of hydrothorax. In ten cases, including the eight fatal cases, there was œdema of the bases of the lungs posteriorly.

Digestive system.—The appetite was usually unimpaired. As before stated, diarrhœa preceded the disease; but when Beriberi had once set in, constipation was more common than looseness of the bowels. Indigestion and flatulency were most common, and frequently caused great distress. Vomiting due to dyspeptic trouble was not unrare.

Obstinate and serious retching preceded death in six of the fatal cases.

The stools were frequently examined for anchylostomata, and thymol was administered to many of the patients. In only three cases were anchylostomata found, and in these there were but few. In three post-mortems made, no trace of this

this parasite was noted, nor did the intestinal mucus membrane show any abnormal sign. That in some cases anchylostomata were present is not to be wondered at, when one considers how frequently orientals are the hosts of parasites and how common a disease anchylostomiasis is among those in this Colony; and I think, therefore, that the helminthic theory of the cause of this form of Beriberi may be dismissed as untenable.

Urinary system.—The urine was usually normal in character. In the four cases of wet Beriberi the amount voided per diem was greatly diminished. In one case it merely amounted to five ounces a day.

Temperature.—Ten of the patients had previously to the diagnosis of Beriberi been treated in Fiji for pyrexia. None had any elevated temperature while under my care. In some of the fatal cases a subnormal temperature seemed to precede death.

Terminations.—Eight of the patients died; and in all death occurred suddenly, and appeared to be due partly to paralysis of the muscles of respiration, and partly to paresis of the cardiac muscle. In six of the cases urgent vomiting preceded death. In all there was difficulty in respiration.

Twelve cases seemed to improve slightly, and twenty-two only showed pronounced symptoms of the disease shortly before their departure from this Colony.

Diagnosis.—Although at first I was certainly in doubt; later on the presence of such symptoms as numbness of the skin and œdema of the knees, tenderness of the calf, and absence of knee-jerks, caused me to form a positive diagnosis of Beriberi, that is, peripheral neuritis, apparently of an epidemic character.

Treatment.—The treatment consisted chiefly of rest and nutritious feeding. Constipation was treated by sulphate of magnesia and p. jalapæ co.

The last-named drug, as well as occasional doses of p. elaterii co., were frequently administered to the patients suffering from wet Beriberi.

Such tonics as strychnine, digitalis, and belladonna were given.

For cardiac embarrassment drastic purgatives, as calomel, or elaterium, or gamboge, were used when the patient's condition admitted it; in other cases nitrite of amyl inhalations, or nitro-glycerine, or hypodermic injections of digitalin were made use of.

Massage and faradisation of the wasted muscles were tried; but the treatment, which, I believe, will turn out to be the only effectual one is, the one I suggested in September, 1894, to the Manager in Fiji of the Colonial Sugar Refining Company, viz., the return of all the Japanese to their own country. This was carried out in February, 1895.

At Nausori, all the patients were kept in one hospital and isolated as far as was possible. Their dwellings were also disinfected by having them washed out with a solution of corrosive sublimate.

Morbid Anatomy.—On account of objections on the part of the Japanese, I was not able to make a post-mortem examination on the majority of the cases, but in three cases I managed to perform modified ones.

In

In all these the cavities of the heart were dilated, especially on the right side. The cardiac muscle showed signs of fatty degeneration. The calf flexor muscles of the leg and the supinator longus of the arm were pale, and showed signs of degeneration In two cases sections of the gastrocnemius, when examined under the microscope, showed granular degeneration, whilst striation was not well marked.

The phrenic and pneumogastric nerves were submitted to a similar examination; and in two instances, the endoneurium and medullary sheath showed signs of destruction, which was most marked near the nodes of Ranvier—the myeline substance being collected in fat-like granules. I was unable to obtain permission to examine the brain and meninges, but from the clinical facts I do not think that they would have shown signs of anything abnormal.

In the intestines no anchylostomata were to be noticed, nor did the mucus membrane show any abnormal signs.

Aetiology or Pathology.

This important matter has of late years been the subject of much discussion. Erni of Sumatra, Kynsey of Ceylon, and Giles in Assam, deeming what they term Beriberi or Kàla-azar to be nothing more nor less than anchylostomiasis, while Pekelharing and Winkler, Kerr, Rhodes, Musso, Morelli, and Vanecke, describe the disease known as Beriberi to be peripheral neuritis of microbic origin. This difference of opinion seems to be due to the fact that the term Beriberi has been applied both to cases of anchylostomiasis and to cases of epidemic peripheral neuritis.

The cases I have ventured to draw attention to in this Paper, certainly seem to belong to the last-named class, the clinical features and the pathological appearances all pointing to such a conclusion.

Among the Indian immigrants here anchylostomiasis prevails to a great extent, and thus the symptoms of that disease and those of Beriberi can be studied side by side. In the one we find anæmia, due to either direct loss of blood caused by the *dochmius duodenalis*, or to septic absorption in the duodenum of materials excreted by these parasites, or, may be, to both causes combined; while in the other, there is *no anæmia* and all the symptoms are those of peripheral neuritis.

The doctrine of epidemic peripheral neuritis being of microbic origin is a favourite one; and, there can be no doubt that the disease can be carried from one place to another by means of human intercourse, and spread when thus carried as, I believe, occurred, in 1891, in New Caledonia, when natives of that island caught the disease from Japanese who came there suffering from it. The disease probably extending by soil or place inoculation. Here, fifty Japanese came to a place where epidemic Beriberi was never known before to exist. They landed in what was supposed to be a healthy condition; and before six months were out forty-two of them had developed the disease, and as the remaining eight had altered electrical reactions they, too probably, would have developed the disease if they had remained here.

This

This place, it is true, would favour the development of the poison, for the climate is damp and the temperature is high, and, perhaps also the diet and the work the Japanese had, predisposed them to the complaint.

Nothing, in the order of the patients attacked, seemed to point to direct personal infection,—those who lived in the same cabin not falling victims to the disease one after the other or at the same time.

The voyage from Japan to Fiji lasted twenty-one days; none complained of Kak'kè, until they had been here two months; thus, if they only received the infection before leaving Japan, this period of incubation would coincide with the time given by Pekelharing and Winkler, who state it may vary from five weeks to several months—the period depending on the idiosyncrasy of the individual and the degree of concentration of the poison.

Remarks.

That the epidemic was confined to the Japanese is, I presume, due to the fact that they were more exposed, as well as more susceptible, to the contagion.

As absence of knee-jerks seems to constitute a premonitory symptom, should the Colonial Sugar Refining Company introduce any more Japanese it might be as well for these immigrants to be medically examined before being admitted to Fiji, and then the presence or absence of knee-jerks, as well as other prodromous indications of Beriberi might be looked for; and in shipping Japanese the examining surgeon might be requested to give particulars concerning pretibial œdema and anæsthesia, as well as tender calf muscles and impaired knee-jerks.

I have, &c.,
CHARLES T. W. HIRSCH,
District Medical Officer, Rewa.

LIST OF ENCLOSURES.

No. 6.

[*Enclosure A.*]

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No. 7.

[*Enclosure B.*]

Famekuso Uchisiba was, during July, 1894, frequently an out-patient for gastric disturbance.

He first came under my observation during August, when he complained of feeling languid and of loss of sensation about the knees.

He stated that he had Kak'kè. He was taken into the hospital for observation.

25th August, 1894.—Patient is a well-built muscular man. Heart and lungs, normal; liver, dulness not increased; spleen not felt. Says that in Japan he had always been healthy. Slight loss of sensation over both knees, which are œdematous; knee-jerks both absent; no ankle clonus.

28th August, 1894.—There are absolutely no signs of anæmia. Thymol, in 30-grain doses, was given, fasting, every hour for four hours, but no anchylostomata came away. Blood, under $\frac{1}{5}$-inch objective, showed no indisposition of corpuscles to arrange themselves in rouleaux, and neither excessive granules, crenated corpuscles, nor microcytes. Considerable number of specimens were examined, and in one stained with fuchsin, I thought I detected some very small rod-like bodies.

12th September, 1894.—Patient still complains of anæthesia of skin about knees and ankles, also of stiffness in calf muscles, and occasional severe paroxysms of spasm of these muscles. The knees are slightly puffy, but there is no other œdema; knee-jerks absent; cremasteric and epigastric reflexes present; the muscles of the calf respond more readily to the galvanic than to the faradic currents; pulse rapid; no cardiac bruits heard.

28th September, 1894.—Patient complains of frequent attacks of dyspnœa; œdema about knees has not extended, but the area of anæsthesia seems greatly to have increased, extending from the front of the legs, where it started, downwards to the dorsum of the feet and upwards along the inner surface of the thigh; temperature is normal; occasionally subnormal; suffers frequently from attacks of epigastric pain, especially after a meal; is taking ordinary Japanese diet. P. jalapæ. co. ʒj in the morning when necessary, also half-dram doses of sulphate of magnesia with five minims of t. digitalis, three times a day. Mild currents of electricity are applied to the muscles of the leg and thigh, and massage is also daily employed.

5th October, 1894.—Pulse irregular, rapid, and much accelerated by the slightest effort; anæsthesia has now extended to the soles of the feet and inner surface of the wrists; wrist-drop is to be noticed; muscles of the calf, flexors of the leg, and supinator longus, show signs of the reaction of degeneration. Nitrite of amyl inhalations used when cardiac dyspnœa is urgent. Patient is quite unable to walk, but can swallow, and takes his food well.

20th October, 1894.—Patient continues much the same, the only difference being that the skin of abdomen is anæsthesic, and attacks of dyspnœa are more frequent and also more severe.

24th October, 1894.—Diet has been increased by the daily addition of two ounces of whiskey and two eggs. Faradisation and massage is continued, and medicine has been changed to a tonic consisting of five grains of ferri et ammon cit with five drops of liq. strych.

3rd November, 1894.—Patient totally unable to walk; attacks, cardiac dyspnœa very severe. Hypodermic injections of $\frac{1}{100}$-grain digitalin used when necessary. Patient taking caff. cit. gr. iii, *2dis horis*.

12th

12th November, 1894.—Patient, who looks much thinner, has now developed aphonia; œdema has not extended; pulse dicrotic.

17th November, 1894.—Patient died suddenly to-day. Seemingly from cardiac paralysis.

No. 8.

[*Enclosure C.*]

S. D., Japanese immigrant, aged 23, came to hospital in August for pyrexia; and, again in October, 1894, was under treatment for the same disease. Was readmitted for œdema and anæsthesia of knees in December.

22nd December, 1894.—Lungs normal; area of cardiac dulness not enlarged; slight tenderness over spine; complains of cramp of muscles of calf; temperature normal; muscles of calf react more rapidly to galvanic than faradic current; knee-jerks absent; no ankle clonus; anæsthesia of knee; slight puffiness about knees and ankles. Patient on ordinary diet, with whiskey 1 oz.

1st January, 1895.—Area of anæsthesia and œdema is not extended; pulse rather rapid; complains of slight attacks of dyspnœa; muscles supplied by peroneal nerve react more readily to galvanic than faradic currents. Patient taking digitalis and mag. sulph. mixture.

11th January, 1895.—Patient continues just the same.

14th January, 1895.—Area of anæsthesia slightly less; knee-jerks absent; has had no attacks of dyspnœa since 1st January, 1895.

1st February, 1895.—Patient left in s.s. "Afghan" for Japan to-day. When seen on the 28th January, 1895, his condition had not altered, and there was neither improvement nor increase in his symptoms.

No. 9.

[*Enclosure D.*]

Daily Diet of Japanese at Nausori:—

Rice	2 lb.
Maibasi	3 oz.
Salt	A sufficiency.
Shoyu	3 oz.
Tea	A sufficiency.
Meat—fresh or tinned, *or* fish—salted or tinned	½ lb.
Hisike, Mis, Sou	A sufficiency.
Fresh lime-juice	2 oz.

No. 10.

No. 10.

Report on an Epidemic of Beriberi that occurred during 1894 in the Labasa District.

District Medical Officer, Henry Noble Joynt, M.A., M.D., D.S.H., to The Honourable the Chief Medical Officer.

Sir, Labasa, 27 May, 1895.

I have the honour to submit the following Report on the outbreak of Beriberi amongst the Japanese coolies working on the Labasa Sugar Estate during the year 1894 to your notice.

I.—History.

In 1894, the Colonial Sugar Refining Co. (Limited), tried the experiment of introducing Japanese coolies into Fiji, for the purpose of working on their sugar-cane plantations. To Labasa were allotted two hundred and five men, to Ba fifty, including two inspectors. The Labasa allotment arrived on April 28th, 1895, and with but few exceptions were a fine body of men. They were located on the Wailevu estate, some three miles from Labasa. This estate had only recently been taken in hand; about half of it had been cleared; and on arrival the Japanese were employed on clearing the unreclaimed portions and draining those already cleared. As a consequence, during their first month's residence fifty were admitted into hospital, of whom twenty-four had malarial fever, ten diarrhœa, and one dysentery. Becoming acclimatised, during June, July and August—three dry, cool months—sickness greatly decreased; the hospital admissions fell to 19, 18 and 13 respectively, and fever, diarrhœa, and dysentery, practically disappeared. On May 30th the first case of Beriberi was admitted into hospital—though not then recognised—during July two, and during August one, similar cases were admitted. They were diagnosed as anæmia or rheumatism, and treated as such. All, with one exception, improved and returned to work. My ignorance of this disease, and the difficulty of obtaining information with regard to symptoms—the interpreter understanding a few words of English merely, and the major part of our conversation being carried out by signs, will account for the non-recognition of the Beriberi. The one exception, mentioned above (No. 798), was admitted on July 9th for an ulcer on his leg. He rapidly became anæmic, developed general œdema of body, and ascites, and died suddenly on August 1st. His symptoms were considered due to a fatty and dilated heart, and the autopsy revealed only the morbid naked-eye conditions which might be caused by such a cardiac lesion. Early in September the men began to complain of numbness and œdema of legs, &c., in larger numbers, which they called 'Kak'kè,' and then naturally Beriberi was recognised. Sixteen cases developed the initial symptoms during the first fortnight of September. On the 14th September, forty-five Japanese and an inspector arrived at Labasa from Ba, of whom nine had well-marked Beriberi, and two remittent fever. Of these three died within a fortnight of arrival. In all fifty-five developed symptoms of Beriberi during September, including four of the non-affected Ba transfers. During October, the attacks per week numbered 19, 29, 24, and 22 respectively. No less than sixty-six more were attacked during the first two weeks of November, by which

which time 219 out of the 250 were suffering from Beriberi. Only five cases occurred during the remainder of November—one in December—and the last on the 20th of January, 1895. The total number of cases recognised amounted to 226. On February 4th, 1895, the survivors, 181 in number, were embarked on the s.s. "Afghan" for repatriation.

II.—General Facts.

Residence.—The Wailevu plantation contains about 680 acres. It is a long triangular alluvial flat, bounded to the north by the sea and a belt of mangrove-swamp of from 2 to 4 or more miles in breadth. Landward it is limited by a low range of reed-clad barren hills. The river Wailevu forms the western boundary; on the other bank of which extends a flat with similar topographical relations. The flat is low-lying and marshy, and was covered with coarse grass, reeds, and pandanus trees. The *lines* stood on a gentle eminence of porous red soil at the base of the hills, were well drained, and kept in good sanitary condition. The houses 70 ft. long by 16 ft. broad, with deep verandahs, were built of wood, raised on piles, wooden-floored, and freely open to the air on all sides. Each house was built to accomodate forty men, allowing 336 cubic feet per man; but the average number of inmates rarely exceeded thirty-two. Each was divided by cross corridors into four compartments, to hold ten men apiece. The woodwork was very rough, most of the timber being unplaned. Although freely opened to the winds it will be seen the construction of the houses tended to promote herding together of the inmates, and the rough state of the timber formed excellent lodging-places for microbes.

The *drinking-water* was of excellent quality; collected from the roofs of the houses, and stored in a series of large, connected, galvanised-iron tanks.

The *labourers* were all young adult males, ranging in age from 18 to 26 or 28 years, and, with few exceptions, strong broad-chested men of fine physique. A large proportion had Beriberi previously at one time or another in Japan—so the inspector informed me. The Labasa men were recruited from the provinces of Hiroshima and Yamaguchi, the Ba allotment came from the province of Wakayama, where Beriberi is said to be more prevalent than in the former provinces. Two inspectors accompanied the men; one of whom had Beriberi some years before, but neither contracted the disease at Labasa.

Habits.—The Japanese were remarkably clean and tidy in their habits. A hot-bath was indulged in by all each evening after work; many men, however, using the same water. A dressing-gown of heavy cotton material was worn very generally during leisure hours, and served as a night-gown as well. Few individuals possessed more than one, consequently these gowns impregnated with germs must have been a principal source of infection. The houses were kept clean, the floors being covered with mats, on which they slept—three or four men often using one mosquito-screen in common. Their meals were taken under the verandahs, but later, when affected with Beriberi, the meals were often eaten in the sleeping-compartments.

Work.—At first the Japanese were employed in clearing the land, uprooting the pandanus trees, &c. A small gang were set to dig drains, but not proving satisfactory

satisfactory workers were taken of and Indians substituted. Later they were employed on the ordinary work of a plantation, ploughing, planting cane, &c.

Food will be noticed more fully in Enclosure D, on Diet-scales.

III.—Prodromal Signs and Symptoms.

Before entering on the subject of the symptomatology of Beriberi, I may mention that considerable difficulty was experienced in accurately eliciting the patients' subjective symptoms, owing to my ignorance of the Japanese language, and want of a good interpreter.

The *first symptoms* usually complained of were:—a feeling of heaviness and weariness in the legs, with 'numbness' of the calf muscles; slight œdema over the crest and inside of tibia; loss of appetite; giddiness; headache; epigastric pain; vomiting, and other digestive disorders. Initial œdema was not always present; in many cases did not appear for several weeks; and in a good many cases was never noted, but this may have been due to lack of observation. A more or less puffy and pasty appearance of the face was often seen, and nearly always slight anæmia of the conjunctivæ and fauces, or pallor about the cheek and eyes. The heart is subject to palpitation, and the pulse may register 80 to 100 beats or more per minute, on the least exertion. Meantime the calf muscles may hypertrophy, and the region of the skin over the gastrocnemius muscle, the front of the tibia and the dorsum of the foot lose their sensibility to touch, so that later the prick of a needle may be unnoticed. The heart sounds become modified,—the first sound becoming prolonged and often rough, and the second sound reduplicated at the base,—and these modification present themselves quite early.

I regret I was unaware of Pekelharing and Winkler's pathognomonic sign of diminution of electric irritability to the faradaic and galvanic currents, and the establishment of partial reaction of degeneration in the muscles, supplied by the external popliteal nerve until too late to test it in the initial stage; but in later stages the reaction of degeneration was marked in the affected muscles in a series of cases in which I tried electric treatment. No fever, so far as I remember, was observed during the early stage.

Plainly and well marked as the above symptoms appear to one who has had experience of this disease, to a tyro they are obscure; and he may well be pardoned if he diagnoses anæmia, rheumatism, or even 'shamming,' especially in sporadic cases.

IV.—General Incidence and Progress.

All cases of Beriberi show the initial signs and symptoms in a more or less marked degree; but, with the onset of fresh symptoms, the clinical aspects of this disease vary so much in each instance, that, for the sake of convenience in description, they may be divided into simple-subacute, œdematous, atrophic, and mixed types. Looking upon Beriberi as a multiple peripheral neuritis, caused by a specific bacterium, as demonstrated by Pekelharing and Winkler, all cases of Beriberi must be regarded as fundamentally the same, differing merely in regard to the intensity of the specific poison, the different nervous areas attached by the organism, and the individual physiological resistence shown by the attacked. I purpose, therefore, to describe

describe briefly the signs and symptoms which are characteristic of each of the types I have mentioned, and then to enter more fully into the symptomatology of Beriberi taken as a general disease, as observed at Labasa.

In the *simple-subacute cases* the initial symptoms increased but little in intensity; the numbness extended a little up the leg; perhaps the hands and fingers became weaker and unable to grasp firmly, and somewhat 'numb'; the reflexes became perhaps impaired, and the gait slightly altered—more often unaffected; the patient became anæmic and disinclined or unable to do hard work; but rarely any pronounced symptoms appeared; and at the date of their departure from Fiji, cases of from two to five months' standing showed little variation from the symptoms they developed during the first four weeks. It seemed as if the individual's physiological power of resistence overcame or at least restrained the morbid inroads of the bacterium and its poison.

In the *œdematous forms* the face is markedly puffy and pasty looking. In advanced cases it assumes a bloated appearance; the submaxillary and parotid glands may enlarge; the natural depressions and contours become effaced; and the subcutaneous tissues of the face, chin, and neck, become greatly swollen and œdematous. The legs and feet also become large and œdematous, which œdema extends to the thighs, abdomen, and thorax and upper extremities, so that the patient presents an enormously bulky, bloated, and unwieldy appearance.

The heart is always dilated, chiefly on the right side; the first sound is prolonged, the second reduplicated at the base, and hæmic murmurs develop. The impulse is diffused. *Pari passu* with the extension of œdema in the subcutaneous tissues, the muscles of the limbs become œdematous and hypertrophied, and are painful on pressure. The sensibility of the surface of the limbs, beginning at the tibial region and extending upwards, is impaired, and the prick of a needle unnoticed. The usual paralyses of the muscles and reflexes appear, though not to the extent observed in atrophic forms; and none of the cases at Labasa, suffering from this type, became completely paralysed.

In the *atrophic forms*, on the other hand, the initial tibial œdema disappears; the muscles atrophy; and the patient becomes thin, emaciated and often completely paralysed. The skin is dry, harsh, and desquamates. The muscles supplied by the external and internal popliteal nerves are first affected, then the extensors of the hands, the supinator longus and the flexors of the fingers and hands. Finally, the abdominal and thoracic muscles are implicated. In these cases the sensory lesions are the most marked. The skin becomes anæsthetic over extensive areas of the lower limbs, arms, and abdomen. Paræsthesia, too, is often marked, especially over the abdomen and legs; and pains in the bones (especially the tibia) are so severe that the patient cannot sleep. The reflexes are almost wholly in abeyance. In the last stage the patient is reduced to a skeleton clothed with a rough, dry, lustreless skin, with paralysed limbs, unable to move in bed without assistance.

In the *mixed cases*, symptoms and signs characteristic of both the œdematous and atrophic types are presented together or in turn. There was generally considerable œdema at first; this improved or disappeared, and was followed by atrophy, or else atrophy was followed by œdema. In a few cases this atrophy was followed by a

third

third stage of general œdema. In one case the patient was at first very emaciated; later he became œdematous, and left the country in this condition.

In the classification adopted above I have left out a form of Beriberi described by most writers under the term '*Acute Beriberi*.' It is thus succinctly described in the article on Beriberi in *Quain's Dictionary of Medicine*: "The symptoms of the acute form are pyrexia; rapid anæmia and anasarca; dyspnœa; dulness on percussion; cough and expectoration; scanty urine; effusion into cavities, plural and pericardial; peripheral paralysis well marked; and great nervous depression." One case (coolie No. 798), noticed more fully under section V.—Terminations, corresponded with the above description. Two other cases, noticed likewise later, might also be placed under the heading of acute; but on reviewing them it will be found that death was due to a sudden extension of paralysis, at an early stage of his disease, to the cardiac or pulmonary nerves, for patients died quite as suddenly in more chronic cases. A few other patients developed rapidly the graver symptoms of Beriberi and yet lingered for a long period. Looking therefore upon these rapidly fatal cases as due to an incidental extension of the disease to the nerves controlling the vital organs, which may occur at any stage, early or late, I have omitted this form from the classification adopted.

Taking up the signs and symptoms *seriatim*, and beginning with the *motor disturbances*, we find the calf muscles generally first affected, then the peronei and the flexors of the feet and toes; in short the muscles supplied by the external and internal poplitcal nerves. The patient in a later stage cannot flex his foot, but nearly always can his knee and hips. In a few cases even this power of flexion was lost. The muscles of the hand and fingers are generally the next group of muscles affected after the flexors of the lower extremity; first the extensor group and the supinator longus, later the flexors, and lastly the triceps. In several cases the interossei were paralysed, and in one case the interossei of the right hand were very early affected, and were the only muscles paralysed. The 'drop-wrist' is very characteristic of the later atrophic stage. In the upper extremity, therefore, the nerves affected are the ulnar, median, and musculo-spiral. The nerve lesions are not necessarily symmetrical: one leg or one hand may retain its motor power longer than the other, nor are the corresponding groups of muscles in each limb paralysed at the same time or to the same extent. Out of 119 cases admitted into the plantation hospital on whom notes were made, twenty-nine had distinct paralysis of the upper limb, whilst a large number of all cases had more or less weakness of grasp, and pain and numbness of fingers and wrist not amounting to actual paralysis. At a fairly early stage the abdominal muscles are more or less painful and affected, but the intercostals seem only to be paralysed in the last stage before death. The laryngeal muscles were affected so as to cause aphonia in three cases, but in a goodly number there was a perceptible change in the voice.

The *patellar reflex* is, as a rule, early impaired, and with the progress of the disease wholly suppressed. This condition, however, is not constant, for several cases with well-marked motor impairment exhibited good patellar reflexes, and in some the reflex was exaggerated. The other reflexes are less often weakened, and only in the later stages. In thirty-seven cases the reflex of the patellar tendon was wholly suppressed. The abdominal reflexes were lost in twelve cases, and the left only

only in a thirteenth; in several it was weak. The cremasteric was lost in six cases; the right only in two others; and in two more it was noted as weak. Ankle clonus was never elicited. In thirty-one of the 119 cases total paralysis of the lower limbs was present, and in seventeen others marked ataxia of gait on walking.

Sensory disorders.—Cutaneous anæsthesia or 'numbness' was constantly present over the calf, ankles and dorsum of feet, accompanied by pain on deeper pressure. Pains in the tibial bones, knees, and thigh, were frequent, and once complained of in the popliteal space. The patient would often run his finger along the course of the internal saphenous vein (or saphenous nerve?) from the thigh downwards when asked if he felt pain. The numbness of the legs was progressive: one day the numbness extended over the calf; later the knees pained; later the patient would say he was numb from the thighs down, placing his hands over the regions in question. Pain and numbness of fingers and wrists was commonly complained of. I regret I did not enter into the cutaneous sensory conditions of the upper extremity. Pain and 'numbness' of the abdominal muscles, with anæsthesia of the skin, and the subjective feeling of hardness were common enough; and pain across the chest was noted fourteen times, though it occurred oftener. Investigation of sensory lesions was however difficult and untrustworthy. Hyperæsthesia, as mentioned before, was occasionally marked. Cramps were sometimes complained of, but I never saw an attack.

Heart.—Palpitation and quickened action of the heart on exertion, together with a lengthening and roughness of the first cardiac sound and reduplication of the second seem to be pathognomic of Beriberi. The cardiac area of dulness is enlarged, and the apex beat diffused, often excited and tumultuous. Occasionally hæmic systolic murmurs were heard. There was nothing characteristic about the pulse, except its varying degrees of quickness.

Nor were there any characteristic *pulmonary symptoms*. Dyspnœa, however, was common in the later stages, and several died from paralysis of either the pulmonary nerves or muscles of respiration. Pain across the chest was present with the dyspnœa.

Digestive organs.—The tongue was usually pale and slightly furred; many cases had a bright red tongue with enlarged papillæ, somewhat like a 'scarlatinal tongue.' Anorexia, flatulent dyspepsia, epigastric pain, often severe and persistent, were frequent. Vomiting often ushered in an attack. Frequent and severe emesis for several days was noted in eighteen cases, usually toward the end. Diarrhœa was common at one stage or another, and often severe. The Labasa cases seemed very susceptible to dysentery or dysenteric diarrhœa without much tormina and tenesmus—thirty-two of the cases suffering thus.

The *liver* was sometimes enlarged, but I rarely remember hepatic tenderness being complained of, although several cases of 'nutmeg' liver were found *post mortem*. Slight jaundice, lasting a week or two, was noticed in seven of the hospital cases and in nine of those at Wailevu.

Urine.—Albumen was not detected in any of the examinations. Dysuria was present in four cases, and retention of urine in three others—the catheter having to be used.

Serous

Serous membranes.—Pleurisy was discovered only four times; pericarditis never during life but invariably *post mortem.* Ascites was noted four times.

The *skin* was always harsh, dry, lustreless, and prone to desquamate in atrophic cases; in œdematous cases it was soft and often shining.

The *mental faculties* did not appear to be impaired, nor was the mental depression mentioned by writers noticed. Perhaps this is due to the light-hearted, good-humoured temperament of the Japanese. Undoubtedly the rapid progress of the epidemic, the number of deaths, and home-sickness, had a depressing effect on the men as a whole, but the actual suffering from the disease did not seem to affect them much individually. Drowsiness and torpor were never noticed.

Anæmia was nearly always present, varying in degree to the severity of attack, and most marked in the œdematous cases. It was never as pronounced as in cases of tropical anæmia amongst the Indian coolies. Microscopic examination of the blood disclosed nothing morbid.

Fever was never seen at the onset of an attack, but occasional sharp attacks, raising the temperature to 102°—103° for a few days occurred during the course of the disease; generally, however, if present, the fever was slight. It was noted in thirty cases; in two it distinctly accompanied the onset of œdema. Some of the sharper attacks may have been ague. Remittent fever of a severe type attacked the Japanese during November, and December, and January, and caused the greater number of the deaths. I find that twenty-nine of the 119 hospital cases suffered, besides several who had not Beriberi. Of these twenty-nine cases, eighteen had Beriberi in the simple-subacute form, of whom eight died.

Amongst *miscellaneous symptoms* were: œdema of penis four times, of scrotum twice; carbuncle of neck once; boils twice; twitching of masseter muscles for a week in one case, and of palpebral muscles in another; hiccough, &c.

V.—Terminations.

Death may be due to paralysis of the heart or lungs from implication of the vagus, phrenic, or nerves of the cardiac plexus; to exhaustion; to bowel lesions; or to intercurrent disease. The gravest feature of Beriberi is undoubtedly the liability to degenerative changes in such vital nerves as those that govern the heart and lungs at any period of the disease. If these nerves an attacked early death may be awfully sudden, and hence such cases have been called acute Beriberi. The four following cases are illustrations of this sudden paralysis.

No. 798—one of the earliest cases of Beriberi at Labasa—was admitted into hospital for an ulcer of the leg on July 9, 1894. A few days later he complained of pains in the legs and epigastrium, and looked anæmic. On the 25th his legs were œdematous, and the œdema rapidly became general over limbs and trunk. At the same time the heart became considerably dilated and weakened in action, and the liver enlarged. He died suddenly on the evening of August 1st after about a fortnight's illness.

No. 714, during the third week of September, developed symptoms of Beriberi of a mild nature, with little œdema. By the 28th October the œdema had vanished, and he appeared to have the disease in the simple-subacute form. That afternoon, however, as the hospital attendant told me, he was seized with sudden 'convulsions,' after

after which he slept, but at midnight succumbed to a second seizure. From post-mortem appearances, death in this case was probably due to asphyxia.

No. 802 developed symptoms of Beriberi about October 27. On November 9 he was admitted into hospital with pains and numbness of legs and abdomen, some œdema of feet and legs, anæmia, and slight diarrhœa. No change for the worse occurred in his symptoms. On December 4 he walked over to the dispensary for his medicine, returned to the ward, and, his companions said, dropped down dead. Autopsy showed sign of pulmonary rather than cardiac paralysis.

No. 816 (admitted September 21), who had Beriberi of the simple-subacute type, with jaundice, was attacked about 4 a.m. on November 18th with dyspnœa, and died before noon.

One other case I should like to mention, on account of its sudden fatality, although it is doubtful if it were Beriberi. At the time I diagnosed and certified sunstroke or heatstroke, but with present knowledge am inclined to think it might possibly have been a case of rapid Beriberi. No. 757 was seized, on the night of August 23rd, with vomiting, 'cramps' in the legs and stomach, and diarrhœa. He is said to have been delirious. He had worked in the field all day and had not complained of any illness. On admission, next day, he was in state of collapse, with quick, weak, bounding pulse (84), half-closed eyelids, pains in the epigastrium and limbs. The diarrhœa and vomiting had stopped, and there was no fever. Next day he remained semi-collapsed, with bounding, throbbing arteries, and blowing systolic murmur, and pains as before. He never rallied, but died of asphyxia on the 29th. Autopsy disclosed a general venous congestion of all organs. The lungs were deeply congested and oozed fluid blood on section; heart dilated, thin-walled R. side, full of fluid blood, valves healthy; large petechial hæmorrhages over walls, along veins, and especially over auricles; pericardium held a little clear fluid; old pleurisy R. lung; liver small, healthy, extensive old adhesions upper surface R. lobe; stomach, congestion of veins, petechiæ thickly marked along veins, no ulceration; pylorus and duodenum much congested and ulcerated; spots of purple congestion along jejunum and ilcum; ecchymoses were also observed numerously scattered over pleura, and peritoneum.

The majority of cases uncomplicated by intercurrent disease died of asthenia.

At Labasa, fifty-seven of the Beriberi cases died—from

Asthenia simply	6	
,, with emesis	2	
,, ,, simple diarrhœa	4	
,, ,, simple fever	3	
	—	15
Emesis		2
Cardiac and pulmonary paralysis		6
Œdema of larynx and general anasarca		1
Enteritis		6
Dysentery		13
Dysentery with remittent fever		3
Remittent fever		11
Total		57

VI.—Diagnosis.

The chief points of diagnosis, as Drs. Pekelharing and Winkler have so ably pointed out, are: (1) Diminution of electric irritability of nerves and muscles, especially those supplied by the external popliteal nerve, to both currents, with partial reaction of degeneration; (2) Heaviness, pain and numbness of legs; (3) Œdema over tibial crest; (4) Palpitation and quickened heartbeat on exertion; (5) Digestive troubles, such as vomiting and epigastric pain. Later come (6) Puffiness of face; (7) Modification of heartsounds with dilatation of heart; (8) Extention of motor and sensory lesions of legs, and extention to fingers and hands; (9) Extension of œdema; (10) Ataxia with impairment of reflexes; (11) Anæmia.

The diseases with which it may readily be confounded in the early stages are anæmia and muscular rheumatism.

VII.—Treatment.

The treatment of Beriberi may be summed up in a few words: removal of the patient from the infected district to a cold climate. All methods of drug or electric treatment are equally futile. Nervine and cardiac tonics may give relief and husband the strength of a patient for a time, but change is the only curative agent. The treatment which afforded most relief at Labasa, was the administration of quinine, iron and strychnine in combination, with arsenic when anæmia was pronounced. Digitalis and caffeine were used when œdema showed itself, and, if in slight degree, the œdema often vanished; but whether *propter hoc* or *post hoc* I cannot say. In the more pronounced forms of œdema digitalis seemed useless. In two cases with general œdema and ascites a mixture containing liq. ferri acetatis, potassium acetate, and liquor strychninæ rapidly got rid of the fluid after digitalis and caffeine had proved ineffectual. A dozen cases were treated by the faradaic and galvanic currents, without result. The digestive troubles,—vomiting, diarrhœa, fever, &c.,—must be treated on general principles. Hydrochloric acid relieved the epigastric pain and anorexia better than alkalies. Good nourishing nitrogenous food I believe to be as efficacious as any drug treatment.

VIII.—Morbid Anatomy.

Post-mortem rigidity was marked. The muscles were dark, thin and wasted on section. In œdematous cases there is naturally intercellular serum.

The *pericardium* almost always held from two to four or more ounces of straw-coloured serous or sero-sanguineous fluid. In two cases adhesions with adherent lymph flakes were found.

The *heart* was invariably dilated and thin-walled, especially on the right side —sometimes very considerably; the muscular tissues pale or dark, and generally soft and friable; the valves were healthy. In one case, however, the heart was only about two-thirds normal size, with thin walls and injected endocardium.

Passive congestion of the *lungs*, with œdema, and patches of collapse or emphysema at their edges was present in almost every autopsy. This congestion was general and not confined to the more dependent parts of the lung as in ordinary hypostatic congestion. Hæmorrhagic infarcts of both lungs were found three times.

Recent

Recent pleurisy was seen four times—three times on the right side, and once on the left. In many cases old pleural adhesions were found. Petechiæ and ecchymoses were often noticed on the pericardium and pleuræ.

The *liver*, as might be expected from a dilated right heart, was generally enlarged, hyperæmic, and in advanced cases in the condition known as 'nutmeg liver.' Only in three cases was it noted as normal. In one œdematous case it was small, pale, fatty, and bile-stained. In two there were perihepatic adhesions—not recent.

The *spleen* varied. In uncomplicated cases it looked normal, but after remittent fever it was dark, enlarged, and soft.

The *kidneys* were often congested; sometimes so as to freely ooze blood on section. The cortex seemed sometimes to be thinner than normal.

Owing to the large number of cases in which *intestinal complications*, such as dysentery, enteritis, &c., were present, it is difficult to say if any of the bowel lesions were due to Beriberi alone; but I am inclined to think not; for in the few autopsies held early in the epidemic on uncomplicated cases, the intestines were pale but healthy. With this statement may be contrasted the appearance in the autopsy on No. 757 detailed above, Section V, who is supposed to have died of heatstroke.

Anchylostoma duodenale was never noticed in any of the autopsies.

To sum up: cardiac dilatation with or without hypertrophy, especially on the right side, seems to be the characteristic naked-eye lesion; all other appearances are due to congestion following this cardiac weakness and obstruction to blood flow, whether it be hyperæmia of the solid organs or effusion into the serous cavities.

IX.—Ætiology.

An exotic disease, imported by and confined to a couple of hundred young male adults, occurring in a strictly localised place like the Wailevu plantation, for a brief period of less than nine months, gives little opportunity to an observer for ætiological investigation. On the other hand these very restricted conditions, by the exclusion of so many heterogenous ones found in countries where Beriberi is endemic, afford an excellent opportunity for confirming the experiences of other observers as to what are the essential factors in its causation. As far as I can trace, the first occurrence of symptoms of Beriberi came under notice about May 30th, that is, one month after the arrival of the Japanese in Fiji. Hence we may conclude the disease was imported either by persons in whom the germs already existed in a dormant state, or else in their clothes and belongings—which seems more likely. That the disease is but slowly infective is shown by the rarity of fresh cases—three only—during the first four months of their residence. It was not until September that the epidemic lit up. The accompanying chart (Enclosure A) shows the weekly incidence of the disease, and the weekly meteorological conditions.

Temperature has a marked influence. In Japan, Kak'kè or Beriberi is only present during the hot summer and autumn months; the advent of cold destroys it. Now Drs. Pekelharing and Winkler found that the Beriberi micrococcus developed best at the temperature of C. 37° (F. 98·6°), but at 25° C. (77° F.) it still grew fairly well.

well. 'It is only below 20° C. (68° F.) that the growth becomes insignificant.' In Fiji, at or about the sea-level, therefore, we have the required heat all the year round. The mean minimum temperature for the three months of June, July, and August, ranged from 68°—71°, in two weeks only falling to 64° and 66°. The mean maximum temperature was 82°—87°. The mean minimum temperature for September, October, and November, ranged from 68·5° to 73°, the maximum from 82°—89°.

Moisture likewise has an important influence. If the rainfall be examined it will be seen that for the ten weeks—June and July to middle of August—only 3·19 inches of rain fell, an amount insufficient for active development; but from August 10—31 as much as 4·26 inches fell. This heavier rainfall was followed by the first outbreak of cases. Again for the two weeks September 14—21, the rainfall rose to 5·87 inches, and during October 5—12, 5·53 inches fell. It will also be noticed that about three weeks after each period of heavy rainfall there was a marked increase in the incidence of Beriberi. The nature of the land was also a factor; the low-lying, alluvial, swampy flats affording a resting-place for the rain and increasing the humidity of the air.

The third essential factor, the *crowding together* of the victims, was also markedly present. The barrack life of the Japanese, and their personal habits of herding together, and sleeping under a common mosquito-screen, as previously alluded to, must have had an undoubted influence in propagating the disease.

Secondary causes, were probably the non-nitrogenous nature of their *food*—the staple diet being rice. Many of the coolies would not eat meat; and the Inspector told me he noticed that the meat-eaters were as a rule the last attacked; certain it is that only one of the cooks contracted the disease. As a rule, too, the weakly and men of low physique were first attacked; but debility and low physique seemed to have little influence on the course of Beriberi when once its progress was established. It was noticed that some of the strongest individuals succumbed the most rapidly.

Messrs. Pekelharing and Winkler say: "It is necessary that a person should have lived for some time in a building or a district where Beriberi is prevalent before he is attacked with the disease." Now neither of the Japanese inspectors, who lived in the European quarters, nor the Europeans who worked with them, and the Indian sick-attendants who looked after them, although they mixed freely with the sick all day, yet slept in different quarters, contracted Beriberi. Nor did any of the Indian coolies, of whom from ten to fifty or sixty lived at Wailevu and inhabited adjacent lines but did not mix with the Japanese in their own houses, contract the disease.

I should like to add a final word with regard to *disinfection* and its efficacy. After the departure of the Japanese, the hospital and lines were disinfected in the following manner:—The interior of the buildings were well washed down with a solution of perchloride of mercury (corrosive sublimate) of the strength of 1 in 1000, to which strong hydrochloric acid was added. Two days later the buildings were well washed with hot water and soap, and left exposed to the air freely for a week. At the end of that period the washings with the corrosive sublimate and soap and water were repeated. The carpenters were then admitted, who altered the buildings so as to suit the requirements of Indian coolies. At date of writing,

none

none of these Indians, after nearly three months' residence, have shown any symptoms of Beriberi, although all the elimatie eonditions—heat and heavy rainfall—have been constantly present. Indeed, the rainfall sinee has been the highest on record; and March and April may be ineluded amongst the hottest months of the year.

X.—Remarks.

In the foregoing pages I have tried to chronicle, as accurately as possible, the aspects of Beriberi as it presented itself at Labasa amongst a comparatively small number of persons. I have approached the subject from a elinical point of view alone, having neither the abilities nor appliances for a more scientific inquiry into its ætiology, pathology, or symptoms. The disease of Beriberi is ignored in the ordinary works which form the library of a medical man, especially on a remote plantation; and I had, therefore, no previous account of the disease on whieh to found observations or follow any partieular line of inquiry. It was not till January, 1895, that Pekelharing and Winkler's work on Beriberi fell into my hands, and enabled me to gain a fuller insight into the disease, and elucidated many of the points which I had up to that period but obscurely grasped. In passing, I may mention that I can heartily indorse the accuracy of the clinical observations contained in this work, most of which, after reading, I can remember to have seen more or less completely in the Labasa eases.

The large number of cases on hand at the same time, the want of skilled assistance, and the serious intercurrent outbreak of remittent fever and dysentery, obscuring and modifying the typical course of the disease, added to one's difficulties. Though no one can be more conseious than myself of its many and grave shortcomings, I trust this Report, which I have the honour to submit to you, may be of some little use, if only as an additional corroboration of facts already noted by much more skilled observers than I can pretend to be.

I have, &c.,

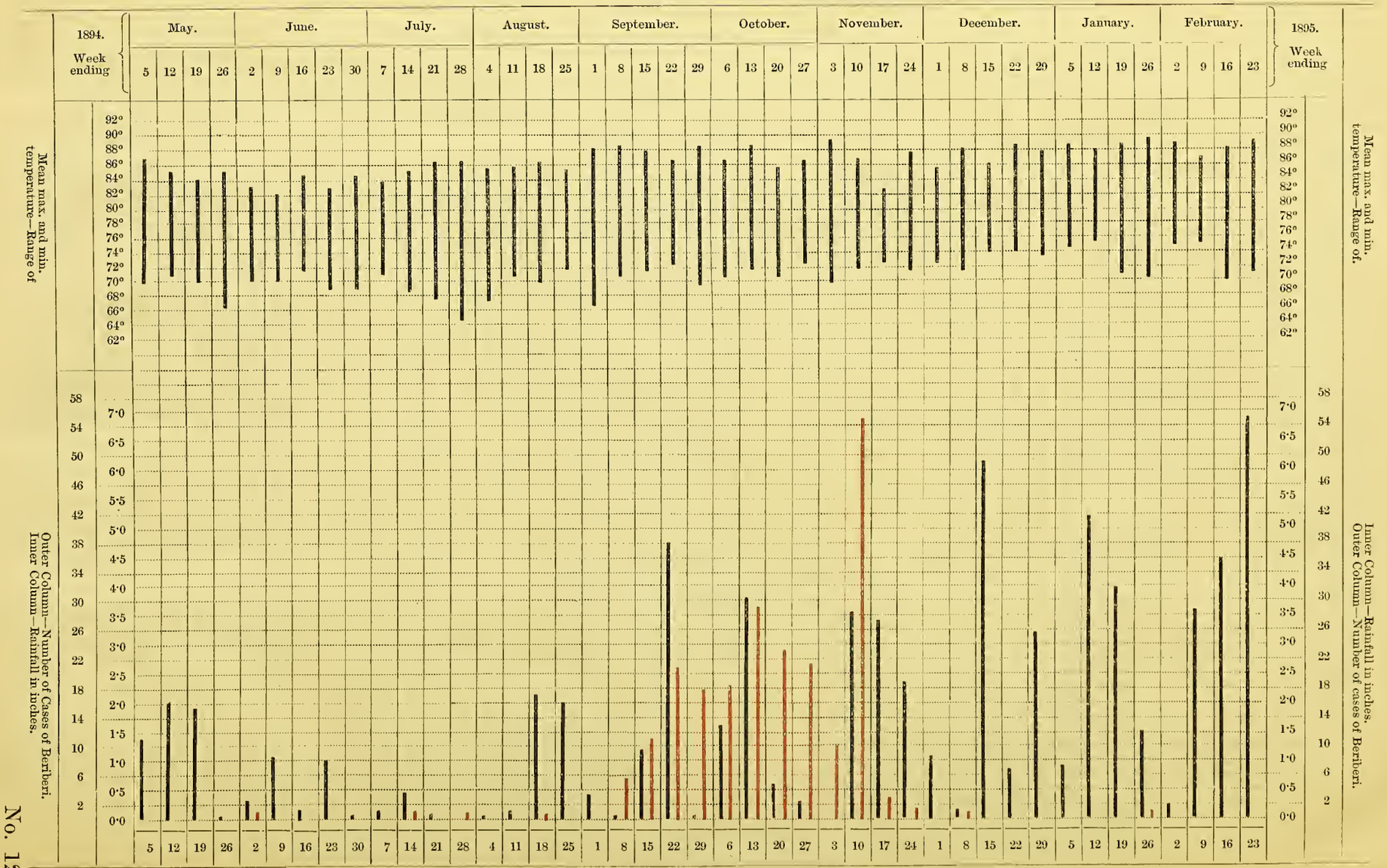
HENRY NOBLE JOYNT,

District Medical Officer, Labasa.

No. 11.

[*Enclosure A.*]

CHART showing range of the Mean Maximum and Mean Minimum Temperature, Rainfall, and Number and Incidence of Cases of Beriberi at Labasa, Fiji, for each week from May, 1894, to February, 1895.



Red lines denote Cases. Lower black lines denote Rainfall: upper ones Temperature range.

No. 12.

No. 12.

[*Enclosure B.*]

HISTORY OF A CASE OF ŒDEMATOUS BERIBERI WHICH TERMINATED FATALLY.

Gazaemon Muraoko (No. 842), was treated at Wailevu, on September 21st, for indigestion; next day he complained of pain across chest; no other symptoms are recorded up to his admission into hospital on October 1st.

His condition then was—slight anæmia; tongue pale and furred; legs numb; slight œdema over tibial crest; pain on pressure; loss of sensation over calf muscles and front of tibia; loss of patellar reflex; abdomen tympanitic; epigastric pain; pain across chest; constipated. He was put on iron, quinine, and strychnine, with hydrochloric acid after meals. He remained in apparently a stationary condition for some weeks. On October 15th he was seized with bilious vomiting, which lasted for four or five days. By the 24th the œdema of the legs had increased; his face was puffy; the anæmia was more pronounced; and he suffered from constant epigastric pain, with tympanitis, and tenderness over abdomen. The leg symptoms remained much the same, but his gait was ataxic. No complaint was made of numbness or pain in the fingers and hands. He was treated with digitalis and iron, and the œdema of legs gradually disappeared.

Early in November œdema of the abdominal subcutaneous tissues and muscles quickly developed. On November 12th the abdomen presented an hour-glass appearance—a fold of œdema extended from the ribs to the navel, and another from the navel to the pubis, with a deep construction between.

There was anæsthesia, and tenderness on pressure; the paresis had increased; the extensor and flexor leg muscles rapidly became implicated; and the patient was unable to walk; the heart was dilated downwards and to the right, the impulse diffused, and the cardiac sounds modified; the face was markedly puffy. On November 28th he had a slight attack of dysentery—temperature rising to 100°—101° —which was checked by ipecacuanha followed by chalk and salol. On December 7th the diarrhœa was better, but the paresis had increased; he was unable to flex his feet; the reflexes, patellar, cremasteric and abdominal, were all lost, and the fingers and hands were numb.

On December 24th, œdema of legs reappeared, and he was put on digitalis. The œdema, however, rapidly increased, extending over legs, thighs, upper extremities, and trunk. On the 31st he was put on acetate of iron and citrate of potash, as the digitalis caused vomiting; but the anasarca steadily increasing digitalis and caffeine were substitued. On January 9th œdema very extensive; slight ascites; loud systolic murmur over base; second sound of heart reduplicated; dysuria; urine scanty, high coloured, no albumen. By the 21st, œdema had reached an enormous extent—he was swollen out like a balloon, enlarged and swollen submaxillary and parotid glands adding to the bloated look of his face; dyspnœa pronounced; slight ascites, but no pleural or pericardial effusion detected; the skin was tense; the voice husky and almost aphonic; the cardiac beat fairly strong; he was unable to walk; could bend his knees and thighs, and had little motor impairment of hands and arms. On February 1st, the anasarca was still increasing; the lungs were œdematous; respiration greatly embarrassed; and œdema of larynx developing. He died that night from œdema of larynx. Autopsy not made. No fever was noted, except during dysenteric attack, but might have been overlooked.

No. 13.

[*Enclosure C.*]

HISTORY OF A CASE OF ATROPHIC BERIBERI WITH TENDENCY TOWARDS RECOVERY (?).

Tsunekichi Kajikawa (No. 788), complained for the first time, at Wailevu, on September 6th, of 'numbness' of legs. On the 11th he again complained of leg-numbness and vomiting, but went to work. On the 14th he ceased working and was sent into hospital next day.

Condition

Condition on admission: slight œdema over tibia, legs numb and weak, no anæsthesia detected; pain in knees; slight ataxic gait; patellar reflex suppressed or else only weak; pains in hands and wrists; weakness and numbness of fingers; face puffy. On the 24th the œdema had left legs, but he complained of much pain in the fingers, hands and arms of both limbs, and across chest; pulse 108; anæmia becoming marked. During the first half of October the motor and sensory lesions became more pronounced; he felt 'numb from the knees down;' and he developed 'drop wrist' from paralysis of supinator longus and extensor muscles. The calf muscles, the peronei group, the dorsal muscles of foot, and extensors of fingers and hands were atrophied; there was pain on pressure. On October 21st there were œdema and anæsthesia over abdominal muscles, and epigastric pain. During November he continued in much the same state; the reflexes had wholly disappeared; he could not flex his feet, but could bend the knee. Emaciation and anæmia were marked. The skin was dry, harsh and desquamating; the usual cardiac symptoms were present. From December 20th—30th he had a mild attack of fever, the evening temperature rising to 100°—102°, the morning temperature ranging from normal to 100°. On January 2nd, 1895, slight œdema of legs was noted, which disappeared in a few days. From this date to his departure, no further development of symptoms were seen, and I find 'improved' marked opposite his case a couple of times, which, however, does not mean that he was tending towards recovery. Possibly the change of climate to a Japanese winter, if he did not die on voyage, might restore him. It would be interesting to know if recovery from paralysis, and to what extent, occurs in these cases on removal to more congenial surroundings.

No. 14.

[*Enclosure D.*]

DIET SCALE OF JAPANESE LABOURERS AT WAILEVU.

Daily rations to consist of:—

Japanese rice (cleaned)	2 lb.
Fish, dried or salted	¼ lb.
Japanese condiments:	
Shoyu	1 lb.
Miso (mashed beans)	
Preserved vegetables (plums, salted) and... ...	
Fresh vegetables or potatoes	
Fresh fish or meat	½ lb.
Japanese tea	½ oz.

This supply of rations to be obligatory for the first six months; after that, food may be gradually assimilated to that of the locality if Japanese Inspector consents—a suitable dietary to be arranged between Inspector and Employer.

These are the articles set down by contract; but in addition to these they used to get sugar and salt, and latterly bread. Of the shoya, miso, and plums, they received ⅓ lb. per head, and of potatoes about ½ lb. They received fresh meat twice a week, but on the other days smoked or salt fish.

Suva: Edward John March, Government Printer.—1896.